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NEW YEAR MESSAGES : NUWEJAARSBOODSKAPPE

FROM DR. J. H. STRUTHERS, PRESIDENT, MEDICAL ASSOCIATION OF SOUTH AFRICA

In retrospect over the past year, the outstanding event in the life of the Association was the South African Medical Congress held in Pretoria in October.

The scientific meetings were splendidly supported by the whole profession and the presence there of medical colleagues from overseas, reading papers in their own fields, added a stimulus to many of the meetings and discussions which made them memorable.

The social events in that crowded week were well attended and were generally most successful. The Doctors' Hobbies exhibition was one of the most interesting ever to have been held.

The Congress developed a spirit of unity and cooperation to a very high degree. The Organizing Committee and their supporting workers were certainly successful in achieving this.

In looking forward to the New Year, I would ask that we determine to take that Congress spirit with us in all the affairs of the Association.

The most important development in the medical life of South Africa in the past year was the incorporation in July of the College of Physicians and Surgeons of South Africa. A great many of our members have assisted in bringing this enterprise to fruition and, as the College goes forward to develop its work and activities, we hope that the Congress spirit will assist in bringing about early practical results and that the College will soon be a living and growing entity in our medical life.

Many problems face us in the coming year; the Federal Council of our Association is discussing and may embark on policies which will affect the medical practice of all of us.

VAN DR. J. H. STRUTHERS, PRESIDENT, MEDIESE VERENIGING VAN SUID-AFRIKA

By 'n terugblik oor die pasverstreke jaar vind ons dat die belangrikste gebeurtenis in die Vereniging se lewe die Suid-Afrikaanse Mediese Kongres was wat in Oktober in Pretoria gehou is.

Die wetenskaplike vergaderings is pragtig ondersteun deur die hele beroep, en die teenwoordigheid van buitelandse mediese kollegas wat referate op hulle besondere gebiede gelewer het, het tot baie van die vergaderings en besprekings 'n stimulus bygedra wat hulle onvergeetlik gemaak het.

Die sosiale funksies in daardie drukke week was flink bygewoon en was oor die algemeen uiters geslaag. Die uitstalling van dokters se stokperdjies was een van die interessantstes wat nog ooit gehou is.

Die Kongres het die gees van eendrag en samewerking tot 'n hoë peil ontwikkel. Die Organiserende Komitee en hul medewerkers het gewis in hierdie doel geslaag.

Met die nuwe jaar voor ons, wil ek versoek dat ons ons sal voorneem om daardie Kongresgees te laat voortleef in al die werksaamhede van die Vereniging.

Die belangrikste ontwikkeling in mediese sake in Suid-Afrika gedurende die afgelope jaar was die registrasie van die Kollege van Interniste en Chirurge van Suid-Afrika in Julie. Baie van ons lede het meegewerk om hierdie onderneming te verwesenlik, en, terwyl die Kollege voortgaan met die ontwikkeling van al sy werksaamhede, hoop ons dat die Kongresgees sal help om eersdaags praktiese gevolge te toon en dat die Kollege binnekort 'n kragdadige en steeds ontwikkelende eenheid in ons mediese lewe sal wees.

Baie probleme staan ons gedurende die komende jaar voor die deur; ons Vereniging se Federale Raad beraadslaag tans oor, en mag beleide onderneem, wat die mediese praktyk van ons almal sal raak.

As we slip into 1956, we remember that our aim is 'the enlargement of man in length of days, in usefulness and in happiness'.

Let us take with us that Congress Spirit of Unity and Cooperation.

I wish the Medical Association and each one of you a very happy and prosperous New Year.

Terwyl ons die jaar 1956 begin, laat ons onthou dat dit ons doel is ‚om die mens in lengte van dae, in nuttigheid en in geluk te verryk'.

Laat ons met ons daardie Kongresgees van eendrag en samewerking saamdra.

Ek wens die Mediese Vereniging en u almal 'n baie gelukkige en voorspoedige Nuwejaar toe.

FROM DR. A. W. S. SICHEL, CHAIRMAN, FEDERAL COUNCIL

As Chairman of Federal Council I take this opportunity of wishing all members of the Medical Association of South Africa seasonal greetings. It is my sincere wish that by loyal membership and cooperation our Branches, Divisions, Groups and individual members during the coming year will continue to promote the interests and maintain the dignity of our Association.

As I am nearing the end of my innings as one of the leaders of the Association I feel it a duty to my colleagues to state very frankly that for some considerable time I have felt greatly perturbed about the way in which we as a profession are tending to drift towards commercialism.

The greater part of the work of Federal Council, and indeed of some of our Branches, is concerned with schedules of fees and contract practice in general. Some at least of our Groups are tending to become, or have become, fee-fixing bodies, to the detriment of the academic and clinical interests which should be the main objective of professional groups.

I have said before and I say again that we as an Association are too prone to adopt policies so rigid that they are in some cases impossible to implement. I refer particularly to the present campaign for open panels. By all means adopt a policy of free choice of doctor; but surely under certain circumstances closed panels are not only reasonable but desirable. Let us rather frame our policies with some measure of elasticity.

With twenty years continuous membership of Federal Council, of which I have been Chairman for half that period, the altered tone of most of our discussions to me is very apparent. We are tending to sacrifice altruism and endangering the dignity of the medical profession by catering mainly for the financial status of the individual.

When in our Memorandum of Association we speak of 'honour and interests' let us be true to our principles and really try our utmost to put the emphasis on honour without sacrificing our legitimate interests. Is it not also in the interests of our members to provide library facilities, insurance against material claims, benevolence, educational grants and scope for the interchange of practices and above all 'to promote the medical and allied sciences'? I realize only too clearly that we must protect

VAN DR. A. W. S. SICHEL, VOORSITTER, FEDERALE RAAD

As Voorsitter van die Federale Raad wil ek van hierdie geleentheid gebruik maak om aan al die lede van die Mediese Vereniging van Suid-Afrika feesgroete oor te bring. Dit is my opregte wens dat ons takke, afdelings, groepe en indiwiduele lede deur lojale lidmaatskap en samewerking gedurende die komende jaar sal voortgaan om die belange van ons Vereniging te bevorder en die eer daarvan te handhaaf.

Aangesien my beurt as een van die leiers van die Vereniging nou sy einde nader, voel ek dat dit my verpligting teenoor my kollegas is om rondborstig te konstateer dat ek vir 'n lang tyd al diep bekommerd is oor die wyse waarop ons as beroep tot kommersialisme neig.

Die grootste gedeelte van die werk van ons Federale Raad, en inderdaad van sommige van ons takke, handel oor tariewe en kontrakpraktyk oor die algemeen. Tenminste 'n paar van ons groepe het al gelde-bepalende liggame geword of is besig om dit te word, tot nadeel van die akademiese en kliniese belange wat die hoofdoel van beroepsgroepe moet wees.

Ek herhaal dat ons, as 'n Vereniging, te geneig is om beleide aan te neem wat so eng is dat hulle in sommige gevalle nie uitvoerbaar is nie. Hier verwys ek in besonder na die huidige kampanje insake ope lyste. Gaan gerus 'n beleid van vrye keuse van geneeshere aan; maar ons moet tog erken dat onder sommige omstandighede geslote lyste nie alleen redelik nie maar selfs wenslik is. Ons moet liewers ons beleide met 'n mate van buigsaamheid opstel.

Met twintig jaar ononderbroke lidmaatskap van die Federale Raad agter die rug—vir die helfte van die tydperk as Voorsitter—is die veranderde stemming van die meeste van ons besprekings opvallend. Ons neig om onbaatsugtigheid te laat vaar, en ons stel die waardigheid van die mediese beroep in gevaar deur hoofsaaklik om te sien na die geldelike posisie van die indiwidu.

Wanneer ons in ons Memorandum van Vereniging van ‚eer en belange' melding maak, laat ons getrou wees aan ons beginsels en werklik ons bes probeer om eerbaarheid eerste te stel, sonder om ons wettige belange prys te gee. Is dit nie ook in belang van ons lede om voorsiening te maak vir biblioteekdienste, versekering teen materiële eise, liefdadigheid, studiebeurse en geleenthede vir praktykomruilings, en boweal, vir die ‚bevordering van mediese en verwante wetenskappe' nie? Ek besef maar te goed dat ons ons lede moet beskerm as hulle bestaan bedreig word, maar ons onderhandelinge in hierdie opsig

our members when their livelihood is threatened, but our negotiations to this end should be more realistic and carried out in the correct perspective.

I make no apology for this frank statement and in wishing you a happy New Year I appeal to one and all to reflect on what I have said.

moet meer realisties wees en moet in die regte perspektief gevoer word.

Ek vra geen verskoning vir hierdie onomwonde verklaring nie, en, terwyl ek u 'n gelukkige Nuwejaar toewens, wil ek op elkeen van u 'n beroep doen om na te dink oor my woorde.

EDITORIAL : VAN DIE REDAKSIE

THE URINE CALCIUM

There seems no reason why all doctors should not be able to conduct a rough bedside test for calcium in the urine as well as, and certainly as easily as, for albumin and sugar. All that is necessary is the mixing of equal quantities of urine and Sulkowitch reagent in a test tube, which is then allowed to stand for 3 minutes. Distinct cloudy suspension indicates a large amount of calcium in the specimen, a haze is normal, and complete transparency suggests a low concentration. The active substance in Sulkowitch reagent is oxalic acid, which forms insoluble calcium oxalate when mixed with any calcium solution, in this case urine. The normal reaction is well simulated by using London tap-water in place of urine, but in many parts of South Africa the much softer water would give a reaction which would represent an abnormally low standard for urine. It is no use testing a dilute specimen of urine—an early-morning (or 24-hour) sample is best.

In conditions in which the urine calcium is altered this test may be helpful. What are those conditions? Abnormally low urinary calcium is found in tetany caused by lowered serum-calcium, as in hypoparathyroidism or osteomalacia, but not in over-breathing tetany, which is the commoner condition. The test is of more value in detecting abnormally high urinary calcium. All patients who develop calcium-containing renal or ureteric stones should be tested. In a proportion the hypercalciuria will be due to hyperparathyroidism; in fact it is the most usual way hyperparathyroidism presents. When hypercalciuria is indicated by the Sulkowitch test, more accurate estimations will confirm it, and a raised serum calcium with a lowering of the proportion of inorganic phosphate in the serum will clinch the diagnosis even in the complete absence of any radiographic changes in the bones and with a normal amount of alkaline phosphatase in the serum.

In our patients with renal calculi, a 24-hour urine-calcium of over 200 mg. is highly suspicious, even on a normal diet. To reduce the calcium intake before measuring the urinary output, although previously recommended by Albright, causes a great deal of trouble, and this procedure can readily be dispensed with, provided simply that the patient to be tested is not taking

DIE URINE-KALSIUM

Daar skyn geen rede te wees waarom dokters nie net so doeltreffend, en sekerlik net so maklik, 'n taamlik juiste siekekamertoets vir kalsium in die urine kan doen as vir eiwitstof en suiker daarin nie. Al wat nodig is, is gelyke hoeveelhede urine en Sulkowitch-reagens, gemeng in 'n proefbuisie, wat dan 3 minute lank moet staan. 'n Duidelik troebel suspensie dui op 'n groot hoeveelheid kalsium in die monster; effense wasigheid is normaal, en algehele deursigtigheid beteken 'n lae konsentrasie. Die aktiewe stof in Sulkowitch-reagens is oksaalsuur wat onoplosbare kalsiumoksalaat vorm wanneer dit met enige kalsiumoplossing gemeng word—in hierdie geval urine. Die normale reaksie word goed nageboots as Londense kraanwater in plaas van urine gebruik word, maar in baie dele van Suid-Afrika reageer die veel sagter water op 'n wyse wat 'n buitengewoon lae gehalte vir urine sou beteken. Dit is nutteloos om 'n verdunde urinemonster te gebruik—die versameling van 24 uur, soggens vroeg geneem, is die beste.

Onder omstandighede waar die gehalte van kalsium in die urine veranderd is, kan hierdie toets nuttig wees. Onder watter omstandighede? 'n Abnormale lae kalsiumkonsentrasie in die urine kom voor by rukkramp veroorsaak deur verminderde serum-kalsium (byvoorbeeld by byskildkliergebrek of beenverweking), maar nie by oormatige asemhalingrukkramp wat die algemeenste soort is nie. Die toets is meer waardevol by die uitkenning van 'n abnormaal hoë konsentrasie kalsium in die urine. Hierdie toets moet uitgevoer word op alle pasiënte wat kalsiumbevattende nier- of ureterstene ontwikkel. Tot op 'n sekere verhouding sal die oormaat kalsium in die urine te wyte wees aan byskildkliergebrek; dit is dan ook die algemeenste manifestasie van byskildkliergebrek. As die Sulkowitch-toets hiperkalsurie aandui, sal dit deur akkurate bepalings bevestig word. 'n Verhoogde konsentrasie van kalsium in die serum, met daling in die verhouding van anorganiese fosfaat in die serum, sal die diagnose onbetwyfelbaar bevestig selfs al is daar geen radiografiese veranderings in die beenweefsel te bespeur nie, en al is die hoeveelheid alkaliese fosfatase in die serum normaal.

By ons pasiënte met nierstene is dit onrusbarend as 'n 24-uur-monster van urine meer as 200 mg. kalsium bevat, al hou die pasiënt 'n normale dieet. Hoewel Albright dit eers aanbeveel het, kan dit baie moeilikheid veroorsaak as die inname van kalsium ingekort word kort voordat dit in die urine gemeet word. Hierdie prosedure kan geredelik afgeskaf word, mits die pasiënt wat getoets moet word nie oormatige hoeveelhede melk, melkprodukte of kalsiumtablette neem nie.

excessive quantities of milk, milk products or calcium tablets.

Some patients with renal stones and hypercalciuria, however, are not cases of hyperparathyroidism. Presumably their tendency to calculus formation is due to an inherent renal hypercalciuria with euparathyroidism—there is a diminished tubular reabsorption of calcium. If this is really so, then 'idiopathic hypercalciuria' may represent an innate renal tubular defect, like renal glycosuria or cystinuria. Apparently not all persons with this abnormality develop renal stones, since in analyses of the urine-calcium output of large numbers of healthy people of different ages a few with very high figures are usually found. Presumably some will be in the process of developing osteoporosis; it is known that an excessive loss of calcium in the urine must continue for 5-10 years before the bones are sufficiently demineralized to show definite radiological evidence of it.

To return to our patients with renal calculi and idiopathic hypercalciuria, it is evident that we should attempt to diminish the tubular concentration of calcium. For this it is recommended that the intake of milk and cheese should be restricted and fluids be drunk in large quantities, particularly at night; and sodium acid phosphate may be given to acidify the urine (ammonium chloride must not be used because the metabolic acidosis so produced will itself increase the calcium output).

Osteoporosis has been referred to. (Incidentally it should be mentioned that by the time the clinical state is obvious the urine calcium may have become normal again.) In the osteoporosis of Cushing's syndrome the urine calcium may be very high. Other conditions in which rarefaction of bone is an outstanding feature need to be considered. In osteogenesis imperfecta the urine calcium is normal since this is a congenital state. This fact may sometimes help in the differential diagnosis in an adult case. In rickets and osteomalacia the urine calcium is excessively low or virtually absent. In infiltrative diseases of bone (secondary carcinoma, myelomatosis etc.) the urine calcium is increased. Incidentally, treatment of carcinomatosis of bone with testosterone tends further to raise both serum calcium and urine calcium. The reason for this is not known. In Paget's disease also the calcium loss in the urine may be considerable and is much increased if the patient retires to bed. For this reason patients with widespread Paget's disease should be kept up and about.

The urine calcium should be assessed in all patients suspected of suffering from sarcoidosis. The high serum calcium and urine calcium which are not infrequently found in this disease create a very serious danger to eyesight (through calcification of the cornea) and to renal function. Cortisone is apparently effective in reducing the calcium levels.

Finally the Sulkowitch test is of great value in regulating the treatment of patients who are receiving large doses of vitamin D or dihydrotachysterol (AT 10)

Somige pasiënte met nierstene en 'n oormaat kalsium in die urine is egter nie gevalle van byskildkliergebrek nie. Vermoedelik is hulle neiging tot steenvorming te wyte aan 'n aangebore nierhiperkalsurie met *normale* werking van die byskildklier—daar is 'n vermindering in die heropname van kalsium in die nierbuisies. As dit werklik die geval is, dan is hierdie ‚idiopatiese hiperkalsurie' miskien 'n aangebore afwyking van die nierbuisies, soos nierglikosurie of sistienurie. Dit blyk dat persone met hierdie afwyking nie noodwendig nierstene ontwikkel nie, want onder ontledings van urine-kalsium by groot aantalle gesonde mense van verskillende ouderdomme, is daar gewoonlik 'n paar baie hoë gehaltes. Vermoedelik is party van hierdie gevalle (van hoë gehaltes kalsium) besig om osteoporose te ontwikkel; dit is bekend dat oormatige kalsiumverlies deur die urine eers na omtrent 5 of 10 jaar die beenweefsel in so 'n mate van minerale berowe dat dit definitief radiologies uitgeken kan word.

Ons keer terug na ons pasiënte met nierstene en idiopatiese hiperkalsurie. Dit is duidelik dat ons moet trag om die konsentrasie van kalsium in die buisies te verminder. Hiervoor word dit aanbeveel dat die inname van melk en kaas ingekort word, baie vloeistof moet gedrink word, veral saans; natriumsuurfosfaat kan toegedien word om die urine aan te suur, maar chloorammonium moet nie gebruik word nie omdat die metaboliese suurvergiftiging wat op hierdie manier veroorsaak kan word op sigself die kalsiumuitskeiding sal vermeerder.

Osteoporose is genoem. (Terloops moet dit gemeld word dat teen die tyd dat die kliniese kondisie ooglopend is, die kalsiumgehalte van die urine alweer normaal kan wees.) In die osteoporose van die Cushingsindroom kan die urine-inhoud van kalsium baie groot wees. Ook moet alle ander siektes waarvan uitdunning van die beenweefsel 'n prominente kenmerk is, in ag geneem word. By osteogenesis imperfecta is die urineinhoud van kalsium normaal aangesien die kondisie aangebore is. Hierdie feit is soms tot hulp in onderskeidende diagnose by volwassenes. By rachitis en osteomalakie is die konsentrasie van kalsium in die urine uitermate laag of so te sê afwesig. Dit is verhoog by insyferende beensiektes (sekondêre karsinoom, verspreide, veelvuldige miëloom ens.). Terloops, behandeling van beenkarsinose met testosteroon is geneig om die konsentrasie van kalsium in beide serum en urine te verhoog—waarom, weet ons nie. Ook in Paget se siekte kan die urineverlies van kalsium aansienlik wees, en kan dit nog verder vermeerder word as die pasiënt bedlêend word. Om hierdie rede moet pasiënte met Paget se siekte op die been gehou word.

Die konsentrasie van kalsium in die urine moet bepaal word by alle pasiënte met sarkoïedose. Die hoë gehaltes van kalsium in die serum en urine, wat nogal dikwels by hierdie siektes voorkom, kan weens verkalking van die horingvlies 'n ernstige bedreiging vir die oë word—en ook vir nierfunksie. Klaarblyklik slaag kortisoon daarin om die kalsiumgehaltes te verlaag.

Eindelik is die Sulkowitch-toets ook van veel waarde om behandeling te reguleer by pasiënte wat groot dosisse vitamien-D of dihidrotagisterol (AT 10) om een of ander rede ontvang (byvoorbeeld vir hardkoppige

for any reason (e.g. resistant rickets, osteomalacia, arthritis, lupus vulgaris, Fanconi syndrome, hypoparathyroidism). An excess of calcium in the early-morning urine must be taken as a danger signal indicating vitamin-D intoxication, and the medicament must be temporarily stopped, or at least reduced. An intelligent patient can do his own testing and dose regulation, much more easily than a diabetic subject can do so with Benedict's reagent and insulin.

rachitis, osteomalakie, gewrigsontsteking, lupus vulgaris, die Fanconi-simptomegroep en vir byskildkliergebrek). 'n Oormaat kalsium in die urine soggens vroeg moet as waarskuwing van vitamien-D-vergiftiging beskou word, en behandeling met hierdie vitamien moet tydelik gestaak of ten minste verminder word. 'n Intelligente pasiënt kan self sy toetse doen en sy dosis reguleer; dit is baie makliker vir hom as vir 'n suikersiektelyer met sy Benedik-reagens en sy insulien.

DIPHTHERIA IMMUNIZATION IN JOHANNESBURG AND BOKSBURG FROM 1935 TO 1955

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It was shown in a previous article[1] that diphtheria morbidity and mortality in South Africa have remained almost unaltered for the last 15 years. In Europeans the diphtheria morbidity is at present 30 times as great as that prevailing in England and Wales. Among 519 hospitalized patients in Johannesburg[2] 75% of the deaths and most of the complications occurred in those below the age of 6 years (recently confirmed by Dubb[3] in a study of a non-European population). About 40% of the patients were admitted so late that antitoxin therapy had no beneficial effect.

I have suggested that at the present time too few children are being immunized in South Africa, but that if the number were increased considerably the menace of a serious outbreak would be reduced and morbidity and mortality would be lowered.[2]

METHODS AND MATERIAL

In the present investigation the distribution of diphtheria immunization among Europeans in Johannesburg and in Boksburg was studied.

The information desired was obtained from 4,066 questionnaires returned from various schools and institutions. In brief, the questions asked were: (1) age, (2) history of diphtheria, (3) immunization, primary course and booster. Non-Europeans were not included in the study because information obtained from them is often unreliable.[4]

Age distribution. As diphtheria in South Africa occurs most frequently in children and adolescents[1,2,3] I decided to examine the age-groups below 20 years of age (with one exception—the Queen Victoria Maternity Hospital). Of the 2,253 forms from Johannesburg, 2,015 came from nursery schools, ordinary schools and institutions dealing with young people, and 238 from the Queen Victoria Maternity Hospital. Only persons under the age of 20 years were questioned in Boksburg.

Socio-economic groups. It was clear that it would be impossible to survey the total European population under the age of 20 years in Johannesburg. Therefore two districts were selected, one of a rather high socio-economic standard and the other of a poor socio-economic standard. This procedure could not be carried out in Boksburg. At the Queen Victoria Maternity Hospital an even distribution was obtained by sending out 50 questionnaires daily the first 5 days of one week, because within this period a cross-section of the mothers of Johannesburg visit the clinic.

Methods of analysis. The subjects were allocated to one or more of 6 groups according to their immunization status:

1. Not immunized (no immunization, or one injection only given more than 6 months ago).
2. Unknown.
3. Immunized, without details.
4. Partly immunized (one injection only given less than 6 months before the investigation started, or 2 injections with an interval of *more than* 6 months between each injection).
5. Primary course (a minimum of 2 injections with a maximum interval of 6 months between injections).
6. 'Booster' (a primary course followed by one or more injections after an interval of more than 6 months).

All forms from persons below the age of 20 years were divided into 5-year age-groups: 0-4, 5-9, 10-14 and 15-19. Firstly the percentages of persons with primary courses and booster injections in each group were calculated, and secondly the percentage who had received the prophylactic injection within the first 2 years of life. The first method of tabulation will to a certain extent reflect the artificial immunity in the population at present.[24] The second one tends to demonstrate the degree of immunity conferred on the children in the early stage of life, when it is of prime importance.

RESULTS

Table I shows the diphtheria immunization in Johannesburg among 2,015 persons under the age of 20 years.

From table I, section A, it is observed that 1·7% have suffered from diphtheria. This does not necessarily reflect the chance of contracting the disease during the first 20 years of life, firstly because the subjects will still be exposed to risk for 0-20 years before they reach the age of 20, and secondly because some diphtheria patients died. Knowing, however, that the diphtheria incidence has remained unaltered

TABLE I. JOHANNESBURG: DIPHTHERIA IMMUNIZATION IN 5-YEAR AGE-GROUPS AMONG 2,015 PERSONS UNDER THE AGE OF 20 YEARS. A=TOTAL OBSERVATIONS. B=HIGHER SOCIO-ECONOMIC GROUP. C=LOWER SOCIO-ECONOMIC GROUP

	A						*B*					*C*				
	Present age						*Present age*					*Present age*				
	0-4	5-9	10-14	15-19	*Total*	%	5-9	10-14	15-19	*Total*	%	5-9	10-14	15-19	*Total*	%
Total number.. ..	181	676	667	491	2,015	—	300	211	201	712	—	376	456	290	1,122	—
Previous diphtheria ..	1	8	16	10	35	1·7	—	2	4	6	0·9	8	14	6	28	2·5
Not immunized ..	8	158	150	87	403	20	23	10	18	51	7·2	135	140	69	344	37
Immuniz. unknown ..	1	10	30	35	76	3·8	5	9	10	24	3·3	5	21	25	51	4·5
Immunized without information	9	16	33	28	86	4·3	6	12	12	30	4·2	10	21	16	47	4·2
Partly immunized ..	7	33	29	38	107	5·3	19	13	17	49	6·9	14	16	21	51	4·5
Primary course ..	156	459	425	303	1,342	66·7	247	167	144	558	78·3	212	258	159	629	56
Primary course in %	85·7	68	64	62	66·7	—	82·4	79·5	71·5	78·3	—	56·3	56·6	55·3	56·0	—
Booster	14	88	114	49	265	13·2	59	50	31	140	19·7	29	64	18	111	9·9
Booster in % ..	7·7	13	17·1	10	13·2	—	19·7	23·7	15·5	19·7	—	7·7	14	6·2	9·9	—

for the last 20 years in Johannesburg[1] and that comparatively few cases occur after the 15th year of life[1,2,3] it would appear possible to estimate the chance of contracting the disease from the incidence in members of the two older age groups. A total of 26 cases occurred in 1,158 observations, a percentage of 2·2, which for the above-mentioned reasons should be considered a minimum figure.

Further it is observed that 20% have never received a primary course, 3·8% did not know whether they were immunized or not, 4·3% stated that they were immunized but were unable to give any particulars (in this study they are incorporated in the non-immunized group as it was felt that a considerable number of them had confused the diphtheria immunization with other immunity procedures) and 5·3% had received one prophylactic injection only. A total of 66·7% had received a full primary course. Their distribution in percentage with regard to the single age-groups and booster injections is presented in Fig. 1.

The primary-course immunization rates in all groups over the age of 5 years were much the same, between 62% and 68%, whereas the youngest age-group shows an immunization rate of 85·7%. Only a comparatively small number had received booster injections, the maximum booster rate in a single group being 17·1%.

This raises the question of the protection afforded during the time when they were exposed to the greatest risk. The percentage of primary courses given before the age of 2 years in each group was calculated (Table II). The total number of observations (1982) is slightly smaller than shown in Table I (2,015) as 37 replies had to be excluded because the parents were uncertain of the actual year the immunization took place (stated as 'in childhood'). However, this will not affect the main result.

Table II, section A, reveals a considerable variation in the time of administration of the primary course in different age-groups. This is presented histogrammatically in Fig. 2.

In the oldest age-group, 11·5% received the primary course before the age of 2 years, as against 84·5% in the youngest age-group. Or, since 1935 there has been a clear and steady tendency towards early administration of the prophylactic. It must be emphasized that the youngest age-group is probably not representative for Johannesburg, since the figures are obtained from nursery schools mainly from the higher socio-economic group. Nursery schools in the lower socio-economic group had compulsory immunization. A survey of them would not reflect the true immunization in this group and could therefore not be studied in this connection.

As already mentioned, the material obtained from Johannesburg could, except for the youngest age-group, be divided up into two main groups: an upper (B) and a lower (C) socio-economic group. These two groups

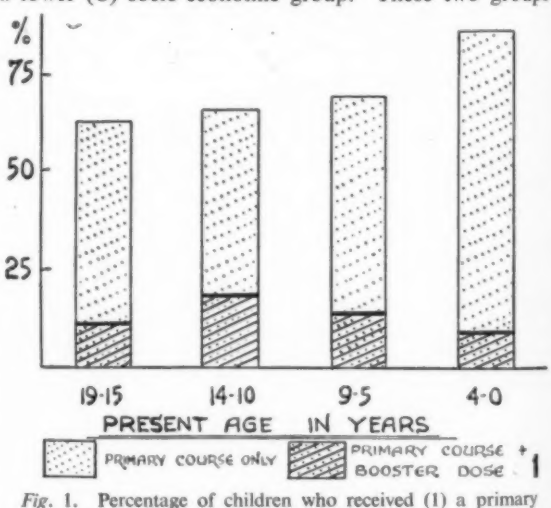

Fig. 1. Percentage of children who received (1) a primary course of diphtheria prophylactic and (2) a booster dose. Johannesburg (total observations 2,015).

TABLE II. JOHANNESBURG: PRIMARY COURSE OF DIPHTHERIA IMMUNIZATION CARRIED OUT UNDER THE AGE OF TWO YEARS IN DIFFERENT AGE-GROUPS

Age	A			B			C		
	Imm. <2 years of age	*Total obs.*	%	*Imm. <2 years of age*	*Total obs.*	%	*Imm. <2 years of age*	*Total obs.*	%
0 – 4..	153	181	84·5	—	—	—	—	—	—
5– 9	330	664	49·9	214	291	74·2	116	373	31·1
10–14	150	653	23·0	88	201	43·8	62	452	13·8
15–19	55	484	11·5	45	199	22·6	10	285	4

Group A, B and C : see Table I. Abbreviations : see Table I.

are compared in Tables I and II. There is considerably less clinical diphtheria in group B (0·9%) than in group C (2·5%). The minimum risk of contracting diphtheria in group B and C during the first 20 years of life is 1·5% and 2·7% respectively. It is worth while noticing that diphtheria has not occurred in the

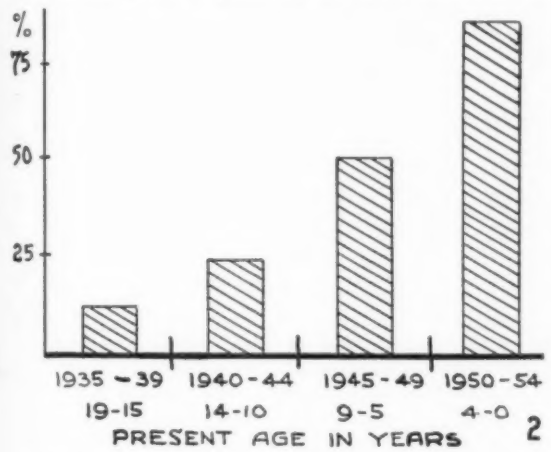

Fig. 2. Immunization rates in different age-groups during first 2 years of life. Johannesburg (total observations 1,982).

5-9 year age-group of high socio-economic standard as against 2·1% in the corresponding age-group of low socio-economic standard. The difference is significant on the 5% level, U = 2·3408.

Further, group B is better immunized (78·3%) than C (56%). The tendency towards increasing prophylactic immunization in recent years of group B is completely absent in group C (Table I). The booster rates are low in both groups, neither exceeding 23·7%, but the percentage boostered in group B is higher than in group C.

The time of administration of the primary course is compared in Table II, sections B and C, and is shown graphically in Fig. 3.

In recent years there has been a tendency to immunize earlier. The proportion of immunized between 1945 and 1949 is more than twice as high in group B (74·2%) as in group C (31·1%).

The analysis of 238 expectant mothers revealed that 5·9% had suffered from diphtheria in the past, 41·2% had not been immunized, 26·1% did not know and 20·2% had received a primary course. None had been immunized or received booster injections during the last 2 years before pregnancy and only 2 within the last 5 years. All that could be ascertained was that in half of them the primary courses had been given more than 10 years ago and in the other half 5-10 years ago.

The immunization rates were also studied in a Reef Town, Boksburg. The results, summarized as in table I, are shown in table III.

About 2% of the questioned persons had suffered from diphtheria. It also appears to be the minimum chance of contracting the disease during the first 20 years of life in Boksburg. It is noticed that 16·2% had not received a primary course, 4·8% were in doubt, 4·5% thought they were immunized but could not give any details and 4% had received one injection only. The remaining 71·4% had received a full primary course. Their distribution with regard to age and booster injections is given in Fig. 4.

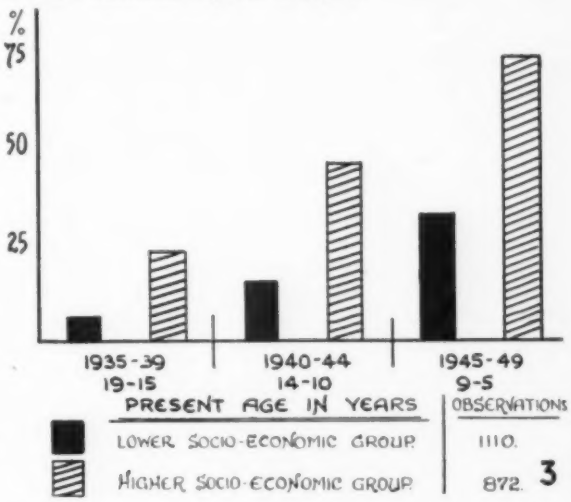

Fig. 3. Immunization rates in different age-groups during first 2 years of life. Different socio-economic groups. Johannesburg.

TABLE III. BOKSBURG: DIPHTHERIA IMMUNIZATION IN 5-YEAR AGE-GROUPS AMONG 1,813 PERSONS UNDER THE AGE OF 20 YEARS

	Present age				*Total*	%
	0–4	5–9	10–14	15–19		
Total number	91	470	912	340	1,813	—
Previous diphtheria	—	11	17	8	36	2·0
Not immunized	32	81	124	56	293	16·2
Immunization unknown	—	14	39	20	73	4·0
Immunized without information	1	21	43	16	81	4·5
Partly immunized	3	17	32	21	73	4·0
Primary course	55	337	674	227	1,293	71·4
Primary course in %	60·5	71·7	73·9	66·8	71·4	—
Booster	—	38	176	42	256	14·1
Booster in %	—	8·1	19·3	12·3	14·1	—

The immunization rates are much the same in all age-groups, between 66% and 73%, except for the youngest age-group where only 60% had received a full primary course. This latter group had not received a single booster injection (91 observations); 8·1% between 5 and 9 years of age had been boostered and the maximum figure of 19·3% was obtained in the 10-14 year group.

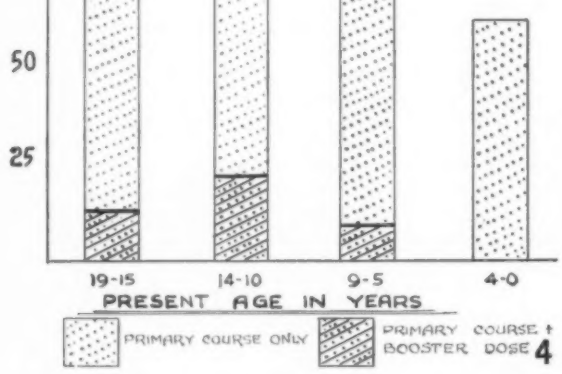

Fig. 4. Percentage of children who received (1) a primary course of diphtheria prophylactic and (2) a booster dose. Boksburg (total observations 1,813).

The number and percentages of persons in each group who had received the primary course before the third year of life are shown in Table IV, and in Fig. 5.

More children have been immunized earlier in recent years: e.g. 6·7% of the 1935-1939 group against 48·4% of the 1950-1954 group, an 8 times increase.

TABLE IV. BOKSBURG: PRIMARY COURSE OF DIPHTHERIA IMMUNIZATION CARRIED OUT UNDER THE AGE OF TWO YEARS IN DIFFERENT AGE-GROUPS

Age	*Immunized < 2 years of age*	*No. of total observations*	%
0– 4	44	91	48·4
5– 9	193	470	41·5
10–14	175	902	19·4
15–19	23	340	6·7

However, the immunization rate of the 1950-1954 group does not lie on the straight line joining the histogram peaks for the 1935-1939, 1940-1944 and 1945-1949 groups. It is lower than it would have been had immunization continued to increase at its previous rate. As a number of the children were about 1 year old they have still a definite chance of being immunized before they reach their second birthday.

Further information, not given in tabular form, showed that 30% of the 5-9, 18% of the 10-14, and 12% of the 15-19 year groups received their primary course between 2 and 5 years of age, which also indicates the tendency to immunize earlier.

DISCUSSION

From the incidence of diphtheria in the two oldest age-groups studied (10-14 years, 15-19 years) it was estimated that the minimum chance of contracting the disease in Johannesburg and Boksburg was between 2% and 2·2%. That this is a minimum estimate is supported by figures from the Queen Victoria Maternity Hospital, where of 238 people interrogated 5·9% had suffered from diphtheria.

The proportion of people who were uncertain of their immunization history increased with age from a negligible figure in the youngest to about 6·7% in the oldest groups. Most of the questionnaires were presumably completed by the parents; but when they were completed by the individuals themselves, as in the case of the expectant mothers, 26·1% could not state whether

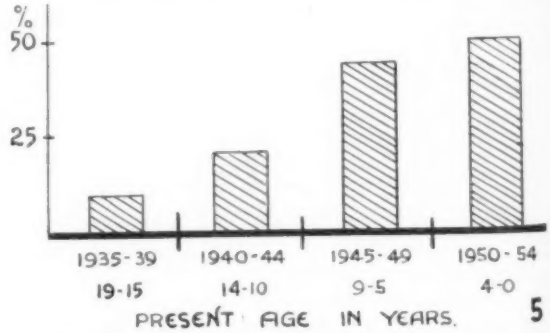

Fig. 5. Immunization rates in different age-groups during first 2 years of life. Boksburg (total observations 1,803).

they were immunized or not. The intelligent use of a vaccination book would leave no doubt on questions concerning immunization in general.

The immunization rates in both Johannesburg and Boksburg in persons under 20 years of age were about 66-72%. Also the booster rates in different age-groups were similar, the average being as low as 13-14% (Tables I and III), which presumably is unique. As the diphtheria morbidity in Johannesburg has remained unchanged during the last 30 years[1] it may be concluded that an immunization rate (primary course) of about 66% (with a variation from 56% in the lower to 78% in the upper socio-economic group) and a booster rate of 15% (variation: 6 to 24%) is insufficient to affect the present morbidity (50 per 100,000 annually[1]). It could be maintained that this is a study of a selected group of the population and that a reservoir of the infection might exist in the non-European population. Infection could spread from them and break through the immunity of the Europeans. That a focus exists in the non-European population was shown by Emmerson[6] and Murray,[7] who found 0·2% and 3·2% carrier rates respectively. It is known, however, that diphtheria epidemics are rare[1] and that the non-European morbidity is lower than that of the European population.[1,7] It would therefore appear reasonable to assume that the non-European population is of less epidemiological significance.

The major reason for the high and constant morbidity is probably the failure to administer prophylactic to the younger age-group,[5,8] an explanation which is strongly supported by the data in Table V. The percentage of immunization carried out under 2 years of age and the total immunization rate is compared with the diphtheria morbidity.

The lack of correlation between total immunization rate and morbidity is striking; e.g. there is not a great difference between the immunization rate in Boksburg and group B, but there is a considerable difference in the morbidity in the 5-9 age-group (B=0%, Boksburg=2·4%). This is probably to be attributed to the higher percentage of children under 2 years of age immunized in group B. The total immunization rate in Boksburg was greater than in group C, yet the morbidities did not differ much. This is probably due to the comparatively small number of children immunized in infancy. It appears that an immunization rate of less than 50% under 2 years of age and a total immunization rate in the older age-groups as high as 73·9% is without influence on the diphtheria morbidity on the Witwatersrand. As the immunization rate under 2 years of age increases, the morbidity decreases (group B). It demonstrates clearly the prime importance of early immunization.[9] *Diphtheria will probably be of insignificant proportions in South Africa when* 75-80% *of the babies are immunized, which is in agreement with observations made in Hamilton.*[10] It can be estimated from Fig. 3 that the 50% mark for the children under 2 years of age in the lower socio-economic group will be reached in 1955-57. Therefore, assuming that the material is representative and that no other factors interfere with the present upward trend in immunization, we should expect that the morbidity in Johannesburg will be reduced in the last years of this decade. The information obtained from Boksburg suggests that the fall in diphtheria in that town can be expected a few years earlier, probably about 1955.

A reduced diphtheria morbidity or, what is the goal, the eradication of the disease from South Africa, could be obtained by the introduction of a compulsory immunization programme for all babies, a procedure current in several countries,[11,12] or through skilled guidance and energetic propaganda for systematic immunization on a voluntary basis. Prophylactic immunization has been advocated often, [13-15,5] and yet for several years ahead we have to face unnecessary deaths and excessive public expenditure due to diphtheria.

It is evident that the primary course of diphtheria prophylactic should be given long before the 3rd year of life. In recent years various studies[16-19] have shown that maternally inherited diphtheria antitoxin in a concentration of 0·1 unit per ml. or more inhibits antitoxin production, whereas 0·04 - 0·02 unit per ml. does not interfere with immunization. It was also found that there was little difference between the antitoxin levels in a group of unimmunized mothers and mothers immunized a long time ago, although there was a considerable variation within the groups.[20,21] Further, 84% of the babies at the 13th week and 96-99% at the 26th week of life had 0·04 unit or less per ml. of serum.[20-22] Therefore, in view of the fact that the majority of the expectant mothers in South Africa are not immunized or were immunized only years ago, it would appear that the optimal time for mass immunization in South Africa would be around the 4th-7th month of life.

When the primary course is followed up by a booster

TABLE V. RELATION BETWEEN THE TIME FOR ADMINISTRATION OF THE PRIMARY COURSE AND THE DIPHTHERIA MORBIDITY

	Johannesburg B			*Johannesburg C*			*Boksburg*		
	Present age			*Present age*			*Present age*		
	5–9	10–14	15–19	5–9	10–14	15–19	5–9	10–14	15–19
Total immunization in % ..	82·4	79·5	71·5	56·3	56·6	55·3	71·7	73·9	66·8
Immunization <2 years in %	74·2	43·8	22·6	31·1	13·8	4	41·5	19·4	6·7
Diphtheria cases in % ..	0	1·0	2·0	2·1	3·1	2·1	2·4	1·9	2·4

Abbreviations : see Table I.

injection 1 year later the child is well protected for at least 5-8 years, independently of the epidemiological environment.[24] It would be wise, however, to give a second booster injection when the child enters school, or at least if an epidemic breaks out more than 5 years after the booster injection.

The prophylactic of choice for the primary course should be toxoid adsorbed on an aluminium-containing carrier such as $AlPO_4$ or $Al(OH)_3$,[25-29] but for the booster it appears that unadsorbed toxoid is slightly superior.[30,31]

Finally it should be mentioned that the association between injection of diphtheria prophylactic and the precipitation of paralytic poliomyelitis, which has been reported when the injections are given intramuscularly,[32-34] has not as yet been observed when the injections are given subcutaneously.[35,36] The latter route, which has been suggested by some workers,[37,38] is the only one used in Denmark.[35,36]

Altogether, there is no justification for the high diphtheria morbidity and mortality in South Africa. At least 75-80% of the parents are willing to have their children immunized. We possess excellent prophylactics. Their appropriate application is the answer to our diphtheria tragedy.

SUMMARY

1. The risk at present of contracting diphtheria on the Witwatersrand during the first 20 years of life is not less than 2·5 - 3·0%. It is highest in areas of low socio-economic standard.

2. Immunization rates vary considerably with the socio-economic standard.

3. Booster rates are unsatisfactorily low in all groups.

4. Immunization carried out under 2 years of age is of capital preventive importance, whereas immunization carried out at a later date, although of benefit to the individual, is of very limited value for the population as a whole.

5. The beneficial effect on morbidity can be expected when a minimum of 50% of the children under 2 years of age is immunized. Eradication of the disease will presumably require 75-80% immunization of the babies.

6. It is estimated that the diphtheria morbidity in Boksburg will decrease from 1955 and that the decrease in Johannesburg will be manifest a few years later, assuming that the present upward trend in immunization continues.

7. The optimum time for commencing diphtheria immunization on the Witwatersrand was found to be the 4th to 7th months of life.

8. The present diphtheria morbidity can be attributed to lack of systematical prophylactic immunization.

I wish to thank Miss E. Bodenstein, Mr. T. Lorimer and Mr. F. Conradie for assistance in tabulating the questionnaires; Mr. N. Berg Sonne, lecturer in mathematics at the University of the Witwatersrand, for valuable criticism; Mr. J. de Bruijne for technical assistance; and Dr. J. F. Murray and Dr. J. Mason for advice.

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MESENTERIC VASCULAR OCCLUSION

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Johannesburg

There are many causes and varieties of acute intestinal obstruction, but none more lethal than that due to mesenteric vascular occlusion. In 1949 Johnson and Baggenstoss[1] wrote: 'Because of the relatively infrequent occurrence of occlusion of a mesenteric vessel, and the poor prognosis usually associated with it, the condition has been largely of academic interest only. Mesenteric arterial or venous occlusion usually has been believed to cause infarction of the bowel and eventual death of nearly all affected persons'. At no period of life, is anybody immune from it. Like a thief in the night, it may strike the healthy, as well as those in the throes of an illness. During the past 10 years I have encountered 9 cases, ranging in ages from 6 weeks to 75 years, with 3 survivals following massive resections. The diagnosis is always difficult; the condition is rarely diagnosed correctly before operation. Sometimes the suspicion of an acute abdomen is not entertained even by experienced practitioners. Surgical treatment, which offers the best and, in the vast majority of cases, the only hope of success, is frequently undertaken too late. It would be better to make no attempt to arrive at an exact diagnosis but to content oneself with the broad diagnosis of 'acute abdomen' and meet the emergency head on without hesitation.

History

Mesenteric vascular occlusion was first described by Antonio Beniviene of Florence in the latter part of the 15th century. It was not until Tiedman and Virchow[2] in the last century described this condition that the medical profession became interested. To Elliot[3] belongs the credit of performing the first successful resection; this was in 1895. Since then occasional reports of single cases and reports from institutions over long periods of time, never amounting to a large series, have appeared from time to time. By 1904, 214 cases of all varieties were reported. Trotter,[4] in an excellent study in 1913, reviewed the literature and reported on 360 known cases. In 1931 Meyer[5] collected 592 cases from the literature. By 1940 Brown[6] was able to find reports on 772 cases. Boyce and McFetridge collected and reported on 13 cases from the records of the New Orleans Charity Hospital over a period of 7½ years. Karn[7] collected 9 cases in a 4-year period at the Dunedin Hospital, New Zealand. In the last series 8 patients were moribund on admission, only one was considered suitable for surgical intervention, and all died. An almost equally high percentage of hopeless cases were encountered by the previous authors. Johnson and Baggenstoss, in a period of 35 years terminating in 1945, reported their autopsy findings in 99 cases of venous occlusion, 60 cases of arterial occlusion, and 12 cases of simultaneous occlusion of arteries and veins. In my own series of 9 cases 3 were moribund when first seen.

Incidence

The incidence has been variously estimated as between 0·10 and 0·20% of all acute surgical cases admitted to any large hospital. Its frequency in cases diagnosed as acute intestinal obstruction varies from 2 to 5%. A correct pre-operative diagnosis had been made in from 4% in Trotter's series to 15% in Cokkinis' series.[8] In the present series of 9 cases, I diagnosed 2 correctly, although in one only did I stand firmly by my opinion (see case reports).

PATHOLOGY

The superior mesenteric artery or vein is usually involved, or any of their branches. Only rarely are the inferior mesenteric vessels affected.

Arterial occlusion may be caused by an *embolus*, which usually arises in the left side of the heart from organized vegetations on the mitral valve and less often on the aortic valve, from a clot in the auricular appendage, or from an organized clot formed on an atheromatous plaque in the aorta. On rare occasions it may be caused by *thrombosis*, resulting from atheromatous changes in the wall of the vessel, or extension of atheroma of the abdominal aorta at its junction with the superior mesenteric artery, or, very rarely, from thrombo-angeitis obliterans of the vessel.

Venous occlusion is always caused by thrombosis, and may be primary or secondary. The former is rare, and may be due to an endophlebitis. The secondary variety is commoner, and is most often the result of acute inflammation arising from any part of the midgut—most frequently the appendix—or from a focus of inflammation arising from the female pelvic organs, and even from infected piles. More rarely it may be due to obstruction of the portal vein from cirrhosis of the liver, pressure of tumours, heart failure, and polycythaemia vera—factors which predispose to thrombosis. There remain a number of cases of venous occlusion for which no known cause can be found; to this the term agnogenic has been applied. In the majority of my cases the cause was never determined.

Whatever the cause, the end result is the same—infarction—to a greater or lesser extent, depending on certain factors. At its onset, the appearance of the intestine at laparotomy differs in the arterial and venous type. In the arterial type, for at least the first 6 hours, the intestine is only slightly dilated and is slightly *cyanotic* in appearance and exhibits movements which may be mistaken for peristalsis, but which are in reality disjointed contractions of separate segments of the bowel. The mesentery at this stage presents a deceptive appearance; on inspection it looks normal whereas careful palpation will reveal that it is pulseless. It is

important to remember this, because in one of the cases (No. 7) it was overlooked, and the intestines were returned to the abdomen, in the mistaken idea that there was nothing amiss, with a tragic result. In both the arterial and the venous varieties haemorrhagic infarction occurs eventually. The magnitude of the infarction will depend on the situation and extent of the occlusion. 'Venous blood can return through a large part of the mesenteric system as long as tributaries are patent, which eventually open into the main trunk above the upper limit of the occlusion, and as long as the anastomosing arcades and radicles which lead from the intestines are patent' (Johnson and Baggenstoss[1]).

In contradistinction to the small intestine and proximal half of the large bowel, the distal half of the large bowel has a much better collateral circulation; hence severe degrees of infarction following occlusion of the inferior mesenteric vein are of rare occurrence. In the rare cases of infarction the intestine and mesentery become swollen, and congested. A haemorrhagic exudate is formed in the bowel wall and lumen and in the mesentery, and sero-sanguineous fluid occupies the peritoneal cavity. As infarction develops the intestine becomes a dull purple colour, loses its glistening appearance and its elasticity and finally becomes soggy, crenated and gangrenous. A septic peritonitis develops. The line of demarcation between healthy and diseased bowel is a gradual one; rarely is it sharply defined. Not only is the affected mesentery friable and swollen but the vessels within it are thrombosed and pulseless. In some cases the infarction is of lesser degree and the haemorrhage may be confined to the mucosa only. This probably occurs in some cardiac lesions that develop abdominal pain and respond to heparin without surgery.

SYMPTOMS AND SIGNS

Whatever the variety the onset is catastrophic, although there are occasional exceptions. These exceptions, few in number, will not be dwelt upon, so as not to make a difficult subject even more difficult; except to state that, in conditions producing grave debility, the symptoms of the vascular lesion are for a period overshadowed by the latter (e.g. uraemia, coma, senile dementia and lupus erythematosus). The patient as a rule is *in extremis* and is beyond the reach of surgery. Generally the picture is that of 'acute intestinal obstruction combined with the signs of internal haemorrhage' as the presenting symptoms. This sums up the broad clinical picture but some details require amplification.

The *pain* is agonizing but is not of a definite colicky nature, and is felt all over the abdomen. *Vomiting* is a feature, but blood in the vomitus is the exception. The patient may pass a few *small watery stools*, and it is exceptional to find blood in them, even on the examining finger, although there may be a large quantity of blood in the small intestine. In one case examination of the stool for occult blood was negative. In one other case (No. 7) was there macroscopic bleeding *per rectum*. Even an enema may produce nothing more than a bloodless stool. *Shock* is profound. The pulse is rapid, and the patient is soon bathed in a cold perspiration. Slowly the abdomen begins to distend, with increasing tenderness to palpation, but there is a characteristic absence of rigidity, although voluntary guarding is present. Bowel sounds, even at first, are not of the tumultous variety; then they fade away to disappear with the advent of paralytic intestinal obstruction. The enumeration of the signs and symptoms is not very helpful, and it is because of this that diagnosis is so difficult.

Radiography is of little help, and should not be relied upon as in ordinary cases of mechanical obstruction.

TREATMENT

Immediate operation is the only treatment. This involves resection of the affected intestine and mesentery and either end-to-end or lateral anastomosis in healthy viable areas, whichever can be effected most expeditiously. This will necessitate keeping wide of the disease where the demarcation is gradual. Even a gravely ill patient may be made fit for surgery by means of a short resuscitative treatment. This will necessitate parenteral therapy for the correction of fluid loss, involving the administration of water, electrolytes and protein in the form of blood, gastric suction for decompression, and sedation for the relief of pain and apprehension. The operator must not be timid, never daunted, but bold enough to remove any length of intestine, even to the extent of all but 18 inches of small intestine, and the right half of the colon. Such boldness may carry with it its own reward. Today with the advent of hibernation anaesthesia, and the general excellence of our anaesthetists, much of the dread of such an operation has been eliminated. The surgery having been completed, the post-operative treatment is carried along the usual lines dictated by circumstances. The pre-operative treatment is carried on with meticulous attention to the maintenance of a correct fluid and electrolyte balance, and every known method of supportive treatment may be invoked.

(*a*) Gastric suction must be maintained and no fluid administered by mouth until bowel sounds are audible or flatus is passed per rectum.

(*b*) Sedatives must now be used sparingly, and especially since the advent of hibernation anaesthesia, the initial doses must be very small, and often, later nothing is required.

(*c*) Hypoproteinaemia, hypovitaminosis and hypokalaemia must be avoided by appropriate therapy.

(*d*) Chemotherapy is employed.

(*e*) Gentle limb and respiratory exercises are instituted. At this stage heparin may be exhibited, but it is not an essential feature of the treatment.

Before concluding my own approach to treatment it is only fair to state that in cases where on laparotomy the degree and extent of the lesion has appeared minimal, closure without resection, followed by anticoagulant treatment, has resulted in a few recoveries (Ochsner). Where anticoagulant treatment has been instituted without laparotomy and a successful result reported, grave doubts as to the correctness of the diagnosis may justifiably be entertained.

PROGNOSIS

When it is so difficult to diagnose a condition, and so difficult to appraise the extent of the lesion pre-operatively, any opinion must be guarded. The prognosis will vary with the general health of the patient and the extent of his vascular disease prior to the onset of the catastrophe, the extent of the lesion, and the distance the pathology has travelled towards its ultimate phase. It is no less dependent on the skill of the surgeon in treating abdominal emergencies. The outlook is grave for, in the best hands, the mortality will be dependent on the duration and extent of the lesion. Thus the lowest reported mortality in 1940 was 60%. Generally it is round about 68% for the venous variety, and almost reaches totality in the arterial variety. In assessing statistics only cases operated upon or autopsied should be accepted. In my cases the mortality was 66·6% in an unselected series.

CASE REPORTS

The gravity of the clinical state of these patients makes it impossible to obtain an adequate history, and it is only in the few that survive that the clinical story can be built up in retrospect.

Case 1

A middle-aged man, admitted to the late Mr. Welchman's ward when I was his registrar. The patient was so shocked that it was difficult to get a history, other than that he had been ill for 4 days with severe continuous abdominal pain, vomiting and inability to pass faeces or flatus. The most notable feature on abdominal examination was a tense, slightly typanitic, very tender swelling, the size of a foetal head, in the suprapubic region unaffected by catheterization. There was nothing else remarkable about the abdomen. Intravenous therapy and gastric suction were started, and when the abdomen was opened, under general anaesthesia, this mass proved to be an enormous caecum. The peritoneal cavity contained a small amount of slightly offensive dark fluid. The entire small intestine, and right half of the colon were black and mostly flaccid, and so were the mesentery of the small intestine and the retroperitoneal tissues in relation to the right half of the colon. The condition was desperate, and I was exhorted by the anaesthetist to close the abdomen. This I did, and the patient died shortly after his return to the ward. Obviously he was almost moribund on admission and nothing would have availed him.

Case 2

S.F., a male aged 40 years, was admitted to a nursing home with a diagnosis of perforated peptic ulcer on 16 April 1944. He was relatively fit except for indigestion, heartburn and flatulence, for which he used to take alkalies, and mild rheumatoid arthritis, for which he took aspirin. He had been well until noon of the day of admission, when after a short stroll he complained of severe generalized abdominal pain, which very shortly became central. He tried to defaecate but passed neither faeces nor flatus and collapsed in the lavatory. On examination at 1.30 p.m., pulse, temperature and respiration were normal and he was not unduly distressed, but felt bilious. He was not restless. Colour good. The abdomen was neither scaphoid nor distended, and moved slightly on respiration. There was but very little guarding, around and above the umbilicus, but the tenderness here was excruciating. Bowel sounds were diminished. Free fluid was not detected. Rectal examination was negative. The blood pressure was not taken. Under general anaesthesia, 4½ hours after the onset, the abdomen was opened. There was a little free odourless blood-stained fluid in the peritoneal cavity. The central portion of the small intestine, and its associated mesentery, presented the typical features of venous mesenteric occlusion. The infarction was irreversible, but had not reached the gangrenous stage. Six feet of jejuno-ileum with its mesentery was resected, and continuity restored by end-to-end anastomosis. The patient left the theatre in good condition and, supported with the usual post-operative treatment, outlined above, he made an uninterrupted recovery. His present nutritional state is very good and he enjoys a normal alimentary life.

Case 3

Mrs. A., a multipara aged 66 years, had been ill for 4 days, with gradually increasing abdominal pain and nausea, followed by vomiting and the passage of irregular small stools until later she could pass neither faeces nor flatus. Only then, on 5 March 1947, did she call in her doctor, who gave her an enema, with a negative result. He diagnosed acute intestinal obstruction and admitted her to a nursing home. There I saw her for the first time. I am given to understand that, before this catastrophe, she had enjoyed good health. She had no cardiac lesions and had never previously been operated upon. The cause of the obstruction was not determined pre-operatively. Being gravely ill, with pallor and other signs of shock, she was given blood, plasma and parenteral fluids, and Wangenstein suction was started. After 3 hours her general condition improved slightly and the abdomen was opened under general anaesthesia, when the diagnosis became obvious. Eleven feet of small intestine was resected, and continuity restored by end-to-end anastomosis. Her general condition remained poor throughout her short convalescence, and she died 3 days after the operation in toxaemic shock.

Case 4

This case is worthy of a detailed description if only to demonstrate the fortitude of an exceptional patient. Mr. S., aged 47 years, was first seen by me at his home on the night of 25 July 1948, his case having been diagnosed by his doctor as a perforated peptic ulcer of 3 hours' duration. His condition was fair, but while his history was being taken he vomited at least 2 pints of stale and fresh blood. He was transferred to a nursing home, given 500 c.c. of blood, and then taken to the theatre, where he received a further 500 c.c. of whole blood during the operative procedure. The peritoneal cavity contained free gas and a fair amount of bile-stained fluid. On the anterior wall of the first part of the duodenum and involving the pylorus was a perforation, ½ inch in diameter and surrounded by a wide circular zone of oedema, infiltration, and fibrosis. The mucous membrane of the posterior wall of duodenum was pouting through the opening. No active bleeding-point was seen. The opening was closed in the usual manner, and the patient was returned to bed with continuous Wangenstein suction and intravenous therapy and fluids. No further bleeding occurred, and all went well until the 4th day, when the suction and intravenous fluids were discontinued, because bowel sounds could be heard.

During the next 24 hours, although he had taken very little fluid, he became extremely distended and commenced to vomit. He was again put on suction, and an enormous quantity of fluid was retrieved, the distension disappeared and his condition improved. It was obvious that he had a complete pyloric obstruction, due to the suturing and superimposed oedema. On the 8th post-operative day he was returned to the theatre and a posterior gastro-enterostomy performed, combined with the Wilson Hay manoeuvre. He made an uninterrupted recovery.

He remained perfectly well until 5 November 1948 when, on his return from a holiday where he had eaten large fatty meals, he developed jaundice. The stools were partly clay-coloured and the urine contained a moderate amount of bile. The liver was palpable one finger-breadth below the costal margin. The gall-bladder was palpable. No vomitus. No pyrexia. On investigation it was decided that the jaundice was of the obstructive type. Ten years before when a cystogram was performed, it was reported that he had chronic cholecystitis without stone. On 22 November he was re-opened. The gall-bladder, was moderately thickened, but not distended, and no stones were palpable. A duodenal ulcer was still present. There were numerous adhesions in the operation area, recent and old, and because of the difficulty and hazard involved in the dissection of the ductal area, a cholecystostomy was performed. The drainage of bile gradually became less, the jaundice subsided, and on the 11th post-operative day a cholangiogram was performed, which showed a patent ductal system with a free flow into duodenum. The tube was thereupon removed. Drainage ceased, and the patient was discharged 4 days later.

On 24 April 1949, after a period of well-being, and while attending a cinema matinée, Mr. S. suddenly felt faint and giddy and vomited copiously. He felt very weak and experienced a little generalized abdominal discomfort. Soon after his admission to a nursing home, the abdominal pain became severe and colicky in nature, occurring in bouts every 3 minutes. He was given a simple enema, and passed a good deal of flatus and some faeces and no blood, after which he felt much better, and wanted to return home. I then saw him at 10 p.m. He appeared comfortable, with normal pulse and temperature and no abdominal distension, but he presented one significant finding: he was a very thin man, and just above and to the left of the suprapubic scar—the relic of the suprapubic drainage of the episode of the perforated peptic ulcer—there was a small localized area of visible peristalsis. The intestine could be seen to rise and fall over an area of 2 square inches, but it was not accompanied by cramps. I thought his trouble was due to a band causing a partial obstruction. Wangenstein suction was applied and intravenous fluids and Omnopon given. He had a relatively comfortable night, but when seen the next morning, the temperature was 99°F, the pulse rate 84 per minute, the abdomen was slightly distended, and his condition had deteriorated. Nineteen hours after the onset his abdomen was opened. The peritoneal cavity contained a small amount of dark blood-stained fluid. The entire small intestine, with the exception of 3 feet from the gastro-jejunostomy to within 6 inches of the ileocaecal junction was infarcted and so was the mesentery. I was able to get well above the upper limit of the infarcted zone, but my judgment was at fault at the lower end. In my attempt to save as much as possible of the intestine, the end-to-end anastomosis was performed with normal intestine above, and what I thought to be viable intestine below, but it was not as viable as I thought. On the 8th post-operative day he developed a small intestinal fistula, which steadily became worse causing a breakdown of the lower half of the wound, and a marked deterioration in his condition. He received all the supportive treatment we knew, but in spite of it he became emaciated, and on 18 May he extruded as a slough what appeared to be the anastomosis, as shown by the row of black interrupted nylon sutures.

On 23 May the abdomen was re-opened. At the site of the anastomosis there was a complete absence of the anterior and lateral walls of the intestine, but the posterior wall was intact and continuous. The opening was closed, with a double row of sutures, an inner continuous No. 00 20-day chromic catgut, and an outer row of interrupted cotton thread. The abdomen was closed with a single layer of thick braided silk. Convalescence was uneventful. His general condition began to improve rapidly. He took his feeds well, and his bowels acted 3-4 times per day. The motion was normal in consistency. For the 9 succeeding months he had about 6 loose yellowish stools daily, occassionally with little warning. Some were of normal consistency. He ate anything and everything. Then for the next year he had 3 relatively normal stools per day, then 2 per day, and since October 1954 he has had one normal motion per day—and no discomfort, no symptoms: he passes flatus easily. He is temperate in his habits, enjoys three meals per day (a heavy meal at night), sleeps well, and has no pain, no heartburn, and very little flatulence. Blood pressure 140/80 mm. Hg. Weight about 138 lb. during the past 3 years. He does a good day's work. The abdomen is well scarred, but shows surprisingly little weakness. There is a small ventral incisional hernia. He does not wear a support.

Case 5

Baby L. O'F.—male, full term, aged 5 weeks—was admitted to the Children's Hospital under Dr. S. Javett on 2 January 1950, desperately ill, and all the worse for a 300-mile car journey. He had seemed perfectly well until 2 days before, when he suddenly passed blood per rectum. Soon afterwards he began to vomit. The vomiting became more copious, and was yellowish, thick and foul-smelling. The stools, which were small and of frequent occurrence, consisted of blood mixed with faeces. The baby had been in obvious abdominal pain since the onset. On examination, temperature 97°F, pulse rate 168 and respiration rate 40. He was pale, shocked, slightly cyanosed, tachypnoeic, and markedly dehydrated, with sunken eyes and a dry tongue. The abdomen was distended and did not move with respiration. Dilated veins were present, coursing over the anterior abdominal wall and lower chest. There was generalized tenderness but little guarding; no masses or tumefactions were palpable, and no free fluid was detected. No bowel sounds could be heard. The rectum was distended, and contained blood mixed with faeces. The pre-operative diagnosis was intussusception. Supportive intravenous and antibiotic therapy was immediately instituted, and the child prepared for operation. On opening the abdomen under general anaesthesia, one was met with a gush of about a pint of dark, foul, thin fluid. The entire small intestine, its associated mesentery, and right half of colon were gangrenous. The abdomen was closed immediately. Death supervened a few hours later. Permission for autopsy was refused.

Case 6

Mrs. G., aged 59 years, a very obese, tall woman, was admitted to a nursing home late at night on 22 September 1951, with signs and symptoms of acute intestinal obstruction of several hours' duration. It was impossible to obtain a clear history, and examination was difficult. A scout X-ray of the abdomen did not give any helpful information. After a short period of supportive treatment she was operated upon under general anaesthesia in the early hours of the morning. The abdomen contained a small amount of sero-sanguineous fluid, and the upper 5 feet of the small intestine from the duodeno-jejunal flexure with the associated mesentery was infarcted. This was resected, and with the greatest difficulty an end-to-end anastomosis in two layers was effected. A Penrose drain was inserted down to the anastomosis. All went well for 7 days, when the drain was removed. The bowels acted, and the patient was taking her feeds well. However, on the 10th day, intestinal contents began to escape from the wound; she developed a generalized peritonitis, and died a few days later.

Case 7

Mrs. A.N.H., a multipara aged 45 years, was admitted to a nursing home as an abdominal emergency at 10 p.m. on 11 October 1951. She was a known sufferer from chronic valvular disease of the heart and fibrillation. A few hours before admission she was suddenly seized with violent generalized continuous abdominal pain. This was soon followed by vomiting and the passage of small bloody stools. Physical examination revealed a severely shocked patient, with subnormal temperature, and a rapid irregular pulse. The tongue was dry, the veins in the neck prominent, the abdomen not distended, and there was generalized abdominal tenderness with no guarding. Hernial orifices negative. On rectal examination no blood on examining finger. A diagnosis of mesenteric thrombosis was made and laparotomy was performed, under general anaesthesia, 2 hours after admission, i.e. 5 hours after the onset of abdominal pain. There was no free fluid in the abdomen, the small intestine was slightly dilated and appeared somewhat cyanosed, the liver and spleen were slightly enlarged and showed evidence of chronic venous congestion and the mesentery appeared normal. The abdomen was closed and the patient returned to her bed. She died the following day. Here were a set of features the significance of which I neither understood nor appreciated. An identical case was reported by Alan A. Klass in 1951.

Case 8

H.W.W., a male aged 60 years, was seen in consultation on 1 February 1953. He had suffered a mild cerebral vascular accident 3 years previously, which left him with a slight residual hemiplegia. About 24 hours before I saw him the attack began with centralized abdominal pain, frequent bouts of bilious vomiting and the passage of small watery stools (macroscopically free from blood). He did not feel very ill and was walking about his home. Little was discovered on examination. The blood pressure was 100/60 mm. Hg, and there was slight tenderness round the umbilicus. He had undergone no previous operations. A diagnosis of partial intestinal obstruction, possibly with mesenteric venous occlusion, was arrived at, the latter in view of his previous cerebral episode. However, a scout X-ray of the abdomen in all positions, a barium enema, a barium meal and examination of the stools for occult blood yielded a negative result. Several days after I had first seen the patient, during which time his condition had not deteriorated, he was given a violent purgative (two No. 9s), unbeknown to me. I saw him a few hours later writhing in agony, with severe centralized abdominal pain, vomiting, and cessation of passage of faeces and flatus.

Laparotomy was performed. The peritoneal cavity contained about a pint of dark blood-stained fluid; in the centre of the small intestines was a 6-foot length of infarcted small intestine, and the

corresponding portion of the mesentery was similarly involved. The affected segment and mesentery was rapidly resected and continuity restored by end-to-end anastomosis. On the 3rd post-operative day he had passed flatus, and was on light diet. The following day his bowels acted with the aid of a mild laxative. He was well on the 5th day. On the 6th day he began to have copious haematemesis, in all probability due to a spread of the thrombotic phenomenon into the splenic vein and thus into the veins of the gastrosplenic ligament. Wangenstein suction, blood and fluids parenterally were given but to no effect, and he died 2 days later.

Case 9

Mrs. M., a widow (multipara) aged 75 years, was awakened from her sleep in the early hours of 1 June 1954 because of the sudden onset of continuous severe upper abdominal and lower thoracic pain. Later she vomited. She passed neither faeces nor flatus from the onset of the attack. Except for constipation for which she had taken laxatives and purgatives over many years, she had always enjoyed good health and was very active for her age. Many years previously she had had an abdominal hysterectomy performed. She was seen 9 hours after the onset by a physician in consultation with her doctor, when she was partially under the influence of morphine. The abdomen was soft and slightly distended, and deep pressure produced some discomfort. The blood pressure was low (100/60 mm. Hg), and the pulse regular but poor in quality. ECG, was within normal limits. The patient was admitted to a nursing home as a case of acute abdominal catastrophe, and was immediately given supportive therapy (blood, fluids and Levophed) because of a deterioration in her condition. Wangenstein suction was started. A series of scout films of the abdomen with a bedside unit were not helpful.

I was asked to see the patient 18 hours after the onset. There was a well-healed mid-line subumbilical scar. She now presented the typical appearance of a combination of acute obstruction and internal haemorrhage, and a pre-operative diagnosis of mesenteric vascular occlusion was made. The pre-operative treatment was continued in the theatre, and she was operated on under hibernation anaesthesia. Fourteen feet of infarcted small intestine, nearly gangrenous, with its associated mesentery was resected, and continuity restored by end-to-end anastomosis. She was returned to bed in good condition. The pre-operative treatment was continued post-operatively until flatus was passed. She made a good convalescence and left for home on the 20th post-operative day with the wound soundly healed and the bowels acting normally. She has remained well, in a good nutritional state, able to resume her normal activities, but still suffers from constipation as before.

CONCLUSION AND SUMMARY

Eight cases of mesenteric vascular occlusion of the venous variety are presented, and one case of embolism of the superior mesenteric artery. The ages ranged from 5 weeks to 75 years. Five were males and 4 females. In only 2 cases was there a possible clue as to the aetiology.

The mortality, including those practically moribund when first seen, was 66·6%. Three were saved, following resections of 6, 12 and 14 feet of small intestine, respectively. Their nutritional state is good, and they lead active normal lives. The first survivor was operated upon 11 years ago, and the last and oldest in the series 10 months ago.

From the foregoing one rightly gets an impression that mesenteric vascular occlusion is indeed a lethal condition. Only timely diagnosis, not necessarily of the exact condition, but rather that of 'acute abdomen', followed by immediate surgery, will lead to happier results.

A great responsibility devolves on the general practitioner not to temporize, but to seek help urgently. The mortality is high and will be lowered only if the time-interval between the onset of symptoms and the execution of surgery, which in itself is relatively simple, is adequately reduced. It is important that the use of chemotherapy, antibiotics and anticoagulants, which all have their uses, should not lead to delay in operating.

It is a sad reflection that of 9 cases, 3 were moribund when seen for the first time. I must take blame for the loss of one of the cases, which could have been saved had I carried out my own message.

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ADDENDUM

Since completing this paper I have seen 2 more cases in consultation. They were males aged 47 and 63 years respectively.

The first was in the last stages of lupus erythematosus, and presented almost unequivocal evidence of an intra-abdominal vascular lesion. No active treatment was undertaken.

The second case started as a cerebral thrombosis. On the 3rd day of his illness, he suffered from severe central and lower abdominal pain, distension, vomiting and inability to pass faeces or flatus. I saw him 48 hours after the onset of the abdominal symptoms, when he was semi-conscious, diagnosed mesenteric vascular occlusion, and suggested that his only slender chance of survival would be a laparotomy. This was performed. The entire small intestine and right half of colon, as I am informed, were gangrenous. The abdomen was closed. He died 2 days later.

CRYPTOCOCCUS NEOFORMANS INFECTION IN BONE

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This report concerns a case of femoral osteitis, from which *Cryptococcus neoformans* (*Torula histolytica*) was isolated. A search through the South African literature reveals 4 papers—Aneck-Hahn (1933), Gray (1940), Sampson and Farren (1942), and Lewin and Roux (1946)—dealing with *Cryptococcus neoformans* infection of the central nervous system in 8 patients, but there is no reference to any case in this country of a definite, localized bone infection, such as is recorded in this paper.

The organism appears to have a predilection for the central nervous system, and osseous lesions are very rare. Collins (1950) finds that in a review of over 200 reported cases of cryptococcosis, only 17, to which he added a further 3, showed bone involvement, the first being that of Busse in 1894. Of this case Collins states: 'Despite this historically important case, bone involvement in torulosis has been recognized only infrequently and, in fact, the absence of lesions in skin and bone was a differential point in first separating this entity from other fungous infections on clinical grounds.'

CASE HISTORY

The patient a Native female aged 9 years, was first seen at the Livingstone Hospital, Port Elizabeth, on 24 April 1955. She complained of pain in the lower part of the right thigh, which had been present 10 days and was associated with a limp and swelling. She had a slight cough and anorexia but no history of fever, focal sepsis or trauma was elicited. On examination a tender swelling apparently arising from the right lower femur was found. Apart from tenderness the usual signs of inflammation were absent.

Radiological examination showed central osteoporosis of the lower femur with erosion of the cortex and elevation of the periosteum. The provisional diagnosis was bone abscess of the right femur, and arrangements were made for surgical drainage, but the patient refused treatment and was taken home. She returned to the hospital 10 days later, on 5 May. The pain was now worse, the swelling had increased and was very tender to the touch, but there was still no local warmth or redness. The temperature was 98°F and the pulse rate 90 per minute.

On incision a thick, grey, rather gelatinous-appearing layer of raised periosteum was found. When this was incised there was a discharge of thick, yellow pus, which was under considerable tension. The underlying bone was found to be necrotic and very soft. The area was drained and a BIPP plug inserted.

Radiological examination of the femur on 29 May showed large saucerization of the lower end with some activity still present on the anterior aspect. There was also some periosteal reaction along the shaft. Similar examination on 27 July showed no further destruction of bone, the disease appearing to be quiescent.

At the time of writing, 3 months after the onset of symptoms, the patient has made a complete recovery, has discarded her crutches and is walking normally.

Laboratory Findings

A specimen of the pus and necrotic bone drained at operation was submitted to the Port Elizabeth Branch of the South African Institute for Medical Research for bacteriological examination on 7 May.

This specimen was blood-stained and had a thick, gelatinous consistency. Direct examination of a smear stained by Gram's method showed a large number of Gram-positive yeast-like organisms each surrounded by a wide capsule which was Gram-negative and had an indefinite, ill-defined outline. This morphology suggested *Cryptococcus neoformans*. A fresh preparation examined by dark-ground illumination showed ovoid to spherical yeast-like organisms surrounded by a wide transparent capsule. There was remarkably little inflammatory exudate.

The specimen was planted on blood agar, nutrient broth and glucose agar, and after 2 days large numbers of isolated colonies were observed on the glucose agar. These colonies were white in colour, round and raised. On staining they appeared as yeast-like organisms, but the capsules observed on direct examination were absent. Wet preparations made from subcultures showed some initial budding, but neither a mycelium nor spores were observed. After a further 2 days the discrete colonies on the glucose agar had become confluent, covering the entire slope; the growth had a deep cream colour and a gelatinous or mucoid appearance. The organism was also successfully subcultured on Loeffler's serum medium, but failed to grow on either blood agar or in nutrient broth.

Various carbohydrate media were inoculated with the culture and incubated at 37°C. In these liquid media it was observed that growth occurred best at the bottom of the tubes, and, in glucose only, after a few days acid was produced. Neither acid nor gas was produced in sucrose, maltose, lactose, mannite, dextrin, galactose, dulcite, inulin, starch, inosite and xylose after 4 weeks' incubation.

From the microscopical, cultural and biochemical findings it was concluded that the organism was *Cryptococcus neoformans*.

Sensitivity tests. Antibiotic sensitivity tests were carried out on blood-agar plates, employing the usual procedure followed for other organisms, i.e. applying dried, impregnated discs onto stroke cultures. Results showed that the organism was resistant to Penicillin, Streptomycin, Aureomycin, Erythromycin, Chloromycetin and Terramycin. Sulphonamide sensitivity tests, using horse-blood agar plates with the sulphonamide incorporated in the medium, revealed complete resistance to Sulphatriad, Sulphamezathine, and Gantrisin.

Biological test. One c.c. of a heavy saline suspension of culture was injected intraperitoneally into a guinea-pig, which was killed 9 weeks later, when there was no evidence of infection.

The patient's *blood count* showed the following results:

	6 *May* 1955	16 *May* 1955
Haemoglobin %	13·2 g.	11 g.
Colour index	0·97	0·90
Red cells per c.mm.	4,650,000	4,090,000
White cells per c.mm.	14,700	18,000
Packed cell volume	38%	34%
Blood-sedimentation rate ..	47 mm.	52 mm.

Occasional red-cells showing increased diffuse polychromasia and punctate basophilia were observed.

Cerebrospinal fluid. A specimen of the cerebrospinal fluid was submitted to check for Cryptococcus infection of the central nervous system. The fluid was clear and without coagula. Culture on various media failed to produce a growth after 15 days at 37°C or at room temperature. Smears of the centrifugalized deposits stained by the methods of Gram and Ziehl-Neelsen showed scanty lymphocytes only. *Cryptococcus neoformans* was not observed.

DISCUSSION

This case presents some interesting features. The fact that the lesion was localized in one bone, and was not a generalized systemic infection, is worthy of note. Cases of cryptococcosis described in the literature show that the parts of the body most commonly involved are the lungs and the central nervous system. To check for such involvement in the present case, radiological examination of the lungs was carried out on 17 May. This revealed no pathological features. Examination of the cerebrospinal fluid (see above) also showed no evidence of cryptococcosis.

The mode of infection is not clear, but it is known that the organism can exist as a saprophyte on the skin, throat and gastro-intestinal tract. It is also found on many plants. Conant *et al.* (1947) describe *C. neoformans* as one of the most inert of the fungi and state that it may exist in the tissues for long periods without causing any inflammatory response. In the present case, however, it seems clear that *C. neoformans* was definitely the aetiological agent of the disease. Leopold (1953) states: 'Torula is rarely a non-pathogenic fungus. Therefore, when this organism is found in sputum, cerebrospinal fluid, in the discharge from an area of bone or joint infection, or elsewhere, with the probable exception of the skin, it should be regarded as the aetiological agent.'

Biochemical reactions are reported as variable and for this reason unreliable for identification purposes. Conant *et al.* (1947) state that many strains ferment glucose only, and this has been found in the present case. Of 2 strains reported by Gray (1940) one was found to ferment glu-

cose and sucrose and the other glucose, sucrose and maltose.

SUMMARY

A case of osteitis caused by *Cryptococcus neoformans* is recorded. The clinical notes and laboratory findings are described and discussed.

I am indebted to Dr. H. van der Post of the Livingstone Hospital for the clinical notes. My thanks are also due to Dr. W. C. Haringston and Dr. F. A. O'Hagan Ward of this Institute for their assistance and advice.

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MILLI-EQUIVALENTS MADE EASY

AN INTRODUCTION TO CLINICAL APPLICATIONS—WITH SPECIAL REFERENCE TO PAEDIATRICS

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'Tell me in milligrams per cent—I cannot understand milli-equivalents.' How often one hears this cry of resigned helplessness when the pathologist's report is read aloud! In point of fact, it is milligrams per cent and volumes per cent that are difficult to understand, because these values are quite irrational and have no bearing on the chemical interrelationship of the blood. But the expression of the concentration of electrolytes in terms of chemical equivalents rather than weight or volume has great practical value. Fortunately the concept of chemical equivalents is not only practical, but is delightfully simple to understand—because it is rational.

Most articles attempting to 'explain milli-equivalents' frighten the prospective reader because the first paragraph or two is usually devoted to a definition of a milli-equivalent. Then the reader beholds in rapid succession mention of atomic weights, equivalent weights, valencies and perhaps millimols; sighs, wonders what medicine is coming to, and goes on to the next paper.

We needn't define a milli-equivalent. It can be understood perfectly well if we know what it does. The term merely indicates an equivalent combining quantity of an ion—not an equal quantity, because the word 'equal' has a connotation of weight—but an equivalent quantity. That is, 1 mEq. of sodium will combine with 1 mEq. of chloride to produce the salt NaCl; *but*, the quantities of sodium and of chloride ion in that salt will not be of equal weight. If these ions could be isolated, and put in separate pans on a balance scale, the chloride ion would be found to be heavier—because it has a higher atomic weight. In the same way, KCl consists of equal mEq. of K and Cl, yet the total quantity of K in that salt weighs more than the total quantity of Cl ion. It is known (and we shall see why later) that 1·0 g. of *NaCl contains* 17 *mEq. of sodium and* 17 *mEq. of chloride*; and that 1·0 g. *of KCl contains* 13·5 *mEq. of each ion*. It will clear be that there are lesser numbers of mEq. making up 1·0 g. of KCl because the molecules of KCl are heavier than those of NaCl. These values of mEq. in 1·0 g. of NaCl and 1·0 g. of KCl should be memorized. They are the easiest means whereby mg.% of K, Na and Cl in those compounds can be converted into terms of mEq./litre.

PLASMA CONCENTRATION OF ION

Now to deal with the normal plasma concentrations of ion expressed as mEq./l. These are shown hereunder:

Cations	*mEq./l.*	*Anions*	*mEq./l.*
Na	142	Cl	103
K	5	HCO_3	27
Ca	5	Proteins	16
Mg + others (Fe, Cu etc)	3	Organic acids ..	6
		PO_4	2
		SO_4	1
Total	155	Total	155

The values that are important in everyday practice are those of Na, K, Cl, and the CO_2 combining power (HCO_3), and must be memorized. These plasma 'normals' are not absolute, and vary somewhat, the normal sodium and chloride values varying some 5 mEq. either way of that shown in the figure, and the normal potassium varying from 4·0 to 5·5 mEq./l.; the CO_2 (more correctly the HCO_3) varies from 23 to 30 mEq./l. (in children 20 mEq./l. may be considered a low normal—especially if the blood is not collected under liquid paraffin).

Note that the cations total 155 mEq./l. (range from 150 to 160) and that the anions total the same. There must always be this equality, both in health and in sickness, in order that the pH of the plasma may remain constant. Moreover, in a state of health the anions and cations together always total 310 mEq./l. (range from 300 to 320), but not necessarily during illness, for conditions may arise where electrolytes (mainly Na and Cl) may be lost in excess of plasma water, so that whereas the total anion will still equal the total cation, their sum may be much short of 310 mEq./l. And with predominant loss of water the reverse may occur,

resulting in the state protrayed by that ghastly word—hyperelectrolytaemia. It is important to know the total electrolyte, if only to have an idea whether to repair deficiency with dilute or with concentrated electrolyte solutions. It will be realized that it is not practical to estimate every cation and anion in the plasma. If one has the values for Na and K, one can add to them an estimated 8 mEq./l. to cover Ca and Mg, and thus have the total cation. Similarly with the anions, if one has the values for Cl and CO_2 (HCO_3) one can add an estimated 25 mEq./l. for protein, organic acids, SO_4, and PO_4. The grand totals will give a rough idea of the total serum electrolyte. An even more accurate estimation of anion can be made if, in addition to Cl and CO_2, the protein value is also known, for then there is only an estimated 9 mEq./l. to add for organic acids, SO_4, and PO_4. In general, however, it is unnecessary to estimate the protein, because it stays reasonably constant unless there is a kidney disease or a nutritional disease.

A further word about the plasma proteins: They normally act as anions, though in states of acidosis they can act as cations to a limited extent. The normal concentration of proteins in the plasma is 16 mEq./l. (or 7·0 g.%, expressed in terms of weight instead of equivalent value). If the concentration of total protein is known in grams per cent it may be converted to milli-equivalents per litre by multiplying by the factor 2·4. e.g. a total protein of 6·7 g. in 100 c.c. multiplied by 2·4 gives the equivalent figure of 16 mEq./l.

Here are representative figures (in mEq.) in various conditions. Advantage is taken of the fact that the total cations always equal the total anions (possibly not always true) to estimate the undetermined anions:

	Total Cation	*Cl*	*HCO_3*	*Undetermined Anions*
Normal	155	103	27	25
Diarrhoea (metabolic acidosis)	146	98	10	38
Severe pyloric stenosis (metabolic alkalosis) ..	145	60	45	40
Pulmonary fibrosis of fibrocystic disease (respiratory acidosis)	150	70	35	45
Salicylate intoxication (respiratory alkalosis) ..	155	110	20	25

There is a common misapprehension that when 'the CO_2 is low' an acidosis is invariably present. As can be seen from the above figures, a 'low CO_2' may occur in respiratory alkalosis, and in respiratory acidosis there is a 'high CO_2'. In actual fact, it is only an estimation of the plasma pH that will tell whether a 'low CO_2' indicates acidosis or alkalosis, but this is seldom necessary because, fortunately, the vast majority of electrolyte disturbances fall into the category of 'metabolic', so that one will usually be right in thinking that a 'low CO_2' indicates acidosis.

THE CORRECTION OF ELECTROLYTE DEPLETION

A few thoughts on the correcting of electrolyte depletion: One would imagine that with the knowledge of (say) the plasma sodium, and the total plasma volume (from the formula *plasma volume=25 ml. per lb. body-weight*) one could supply exactly enough sodium to bring a low value to normal. It doesn't work. And the reason is simply that the plasma has a reservoir—the extracellular fluid—which is some 3 times the volume of the plasma. Both have almost exactly the same concentration of electrolyte. The plasma is in fact the mirror of the extracellular fluid and, when a calculated amount of electrolyte is given intravenously, most of it enters the extracellular fluid. Why then, not give 3 or 4 times the calculated (plasma) deficiency? Because clinically it is unnecessary. By the time the calculated plasma-deficiency has been replaced, and perhaps a little more given for the deficiency in the extracellular fluid the patient is usually so much better that the intravenous infusion can be discontinued, and oral absorption will make up any further lack in extracellular fluid or plasma.

A few words about saline: It is often called 'normal' saline—an unfortunate term from the point of view of clinical therapeutics. Its concentration is 0·9 g.%—or 155 mEq./l. of Na and of Cl. (If 0·9 g.%=155 mEq./l., then 1·0 g. of NaCl must equal 17 mEq. of Na and of Cl). Now while its total cation is the same as the total cation of the plasma, and equally so anion, so far as the substances it is supplying (NaCl) is concerned it is grossly hyper-concentrated, especially in chloride (normal plasma chloride, it will be remembered, is about 100 mEq./l., and sodium about 140 mEq./l.). So that, like the Holy Roman Empire, which was neither holy nor Roman nor an empire, *normal physiological isotonic saline is neither normal nor physiological nor isotonic from the point of view of clinical therapeutics.* There are no paediatric indications whatever for using 0·9 saline subcutaneously, and almost no indications for using it intravenously. It should only be used intravenously (indeed, it may properly be given in a more hypertonic solution—up to 3% NaCl) when there is gross deficiency of plasma sodium and chloride (with total cation+anion much short of 310 mEq./l.) with little or no deficiency of water. This state of absolute hyponatraemia and hypochloraemia occurs occasionally in chronic cardiac failure treated by the giving of mercurials and restriction of salt intake. In this state apathy, confusion and delirium may be remedied with intravenous salt.

Saline, then, should not be used in concentrations of more than '2/3rds normal'. Half-normal saline (with 2½% glucose) is an excellent solution for subcutaneous and intravenous use. It supplies about 77 mEq./l. of Na and of Cl—in safe hypotonic concentration. The relative excess of water is excreted while the electrolytes are retained. To this solution other substances may be added if needed; perhaps calcium, or if there is gross acidosis, sodium lactate may be added.

A note on lactate: drug companies produce ampoules of molar (m) and vacolitres of m/6 Na-r-lactate. 'Molar' means that there is 1·0 mEq. of sodium and of lactate in 1·0 ml. of the solution. One litre of molar lactate will therefore contain 1,000 mEq. of Na and of lactate. One litre of m/6 lactate will therefore contain about 167 mEq. of each of these. In terms of weight it may be stated that 1 g. of sodium lactate contains about 9 mEq. of sodium and of lactate. (To those with a mathematical mind mg.% can easily be converted into mEq./l. by means of

calculations involving ionic weight and valency. In practice it is almost never necessary. All the clinician need remember are the values—already stated—for Na, K and Cl). Very rarely, in gross alkalosis, one can add an acid salt such as ammonium chloride. This can be given as a 1·0% or 2·0% solution. One gram of NH_4Cl supplies 18·8 mEq. of ammonium and of chloride. In alkalosis the chloride will be retained while the ammonium will be metabolized and excreted. Generally speaking, however, supplying a substance for influencing the pH is of secondary importance; one must supply electrolyte and the body will deal with the pH. That is why Ringer's and Hartmann's solutions (which contain largely sodium and lactate) are giving way to Darrow's and Butler's solutions—which contain a considerable amount of electrolyte as well as lactate. Half-strength saline then, is an excellent hydrating solution, and may be varied by the addition of other substances to suit the occasion.

Generally speaking, hydration should be fully established and the kidneys functioning before potassium is given intravenously. It should be given in very small quantities and later increased to the daily needs (these values are mentioned presently). Keen judgment is necessary because, on the one hand, in dehydration potassium given intravenously may cause death by cardiac arrest and, on the other, the giving of sodium chloride only will further deplete the intracellular potassium; because the sodium given intravenously goes into the extracellular fluid and then into the cells, as the result of which potassium passes out of the cells into the extracellular fluid and the plasma, some of it then being excreted *via* the kidney; the net result being intracellular loss of potassium from the intravenous administration of sodium.

A warning about subcutaneous infusions: In severe dehydration subcutaneous solutions are dangerous because:

(*a*) If hypotonic, and especially if containing glucose, absorption is very slow and, in fact, before absorption really begins there is first a redistribution of plasma water and electrolyte with respect to the subcutaneous pool. This may cause an initial decrease (absolute or relative) in one or other plasma-constituent, which may occasionally be 'the last straw to break the camel's back'.

(*b*) Much worse is the giving of a hyper-concentrated solution. It always causes initial extracellular, and hence intracellular, dehydration. All paediatricians of experience have seen infants become collapsed after a subcutaneous infusion of 'normal' saline for moderate dehydration—in association with a transient increase in the volume of the subcutaneous pool (e.g. a thigh may go on swelling after the infusion is over).

If oral feeding is impossible or inadvisable, the basic daily parenteral needs for water and electrolyte (not counting any extra for diarrhoea, fistular loss, gastric suction etc.) is as follows:

Water, 150 c.c. per kg. body-weight (i.e. 2½ oz./lb.) per day
Sodium, and also chloride, each, 5-8 mEq./kg./day
Potassium, 2-3 mEq./kg./day

Solutions such as those of Darrow or Butler (which contain Na lactate as well as K) are valuable in the treatment of diarrhoea, which is commonly associated with an acidosis (largely due to loss of Na and K from the bowel), but they should not be used in the treatment of the potassium deficiency of (say) pyloric stenosis, because a metabolic alkalosis is already present; a neutral solution can be used, as follows:

500 ml. of 'normal' saline + 500 ml. of 5% glucose water. The resulting litre of fluid will then contain about 77 mEq./l. of Na and Cl. Add to this solution 2·0 g. of KCl (that is, 13·5 × 2 = 27 mEq.K and 27 mEq.Cl) and we have an excellent 'gastric replacement fluid' containing 77 mEq.Na, 104 mEq. Cl. and 27 mEq.K. This solution can be modified for use in acidotic states by the addition of suitable quantities of sodium lactate, and it will then closely resemble Darrow's solution.

The accompanying diagram (Fig. 1) indicates the electrolyte concentrations in mEq./l. of plasma, 'normal' saline, Darrow's solution, milk, orange juice, and an average watery stool. An interesting feature is the wealth of electrolyte contained in milk. It is not generally

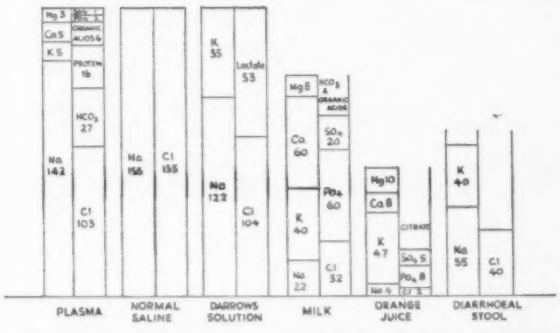

Fig. 1. Electrolyte concentrations in mEq./l. (from diagram in infants' ward, Bellevue Hospital, New York City).

appreciated that milk is an excellent oral rehydrating fluid. When there is no vomiting (with the attendant danger of aspiration) milk would appear to be the best rehydrating and 'electrolyte replacing' fluid. Perhaps the belief that the milk is not absorbed or that 'the bowel needs a rest' needs to be revised. One wonders whether, in the treatment of diarrhoea, the long-standing dictum 'Put him on clear fluids for 24 hours' is really very wise.

SUMMARY

Normal plasma sodium: about 140 mEq./l.
Normal plasma potassium: about 5 mEq./l.
Normal plasma chloride: about 100 mEq./l.
Normal plasma bicarbonate: about 25 mEq./l.

Daily requirements in 'parenteral alimentation':

Water: 150 ml/kg. body-weight/da
Sodium: 5-8 mEq./kg. body-weight/day
Chloride: 5-8 mEq./kg. body-weight/day
Potassium: 2-3 mEq./kg. body-weight/day.
Calories (in infants): 80-100/kg. body-weight/day (if possible)
Vitamins and calcium: q.s.
T.L.C. (tender loving care): q.s.

One gram of NaCl contains about 17 mEq. Na and about 17 mEq. Cl. One gram of KCl contains about 13·5 mEq. K and about 13·5 mEq. Cl.

'Physiological saline' is an unphysiological solution. It should not be used parenterally, unless there is marked loss of plasma sodium and chloride relative to water loss.

In the last analysis it is always the clinical picture that matters. Biochemistry is only an aid, albeit a valuable one. It can be of much assistance in judging what to put in an intravenous solution, but it cannot tell one when to put up a drip, and when to take it down. No amount of investigation can tell one when a drip is necessary, but there is a clinical rule which, when one has had a little experience, is quite invaluable: 'The best time to put up an intravenous infusion is when one begins to wonder if it will be necessary'.

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FAVISM

REPORT OF A CASE OCCURRING IN JOHANNESBURG

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Favism is an acute disorder consequent on ingestion of broad beans (*Vicia faba*) or inhalation of pollen from the flowers. Its occurrence is thought to depend on an allergy in individuals previously sensitized by such contact. In classical form it occurs as an acute haemolytic anaemia.

Children, particularly males, comprise the majority of those affected, and almost all fatalities have occurred in children. The mortality rate is about 8%.

The illness may vary from a severe haemolysis with haemoglobinuria and shock to a milder state in which the attack may cause only malaise, dizziness and slight gastro-intestinal upset, with an increased excretion of urobilin. Intervening forms occur, with pallor, jaundice, vomiting, lumbar pain and weakness.

After ingestion symptoms begin within 5-24 hours. It is commonly the raw bean that is responsible for the illness, although cooked beans have also been implicated.

Symptoms following inhalation may occur with dramatic suddenness within minutes, although more commonly some hours elapse. The inhalation group comprises about 40% of the total number of cases.

The subject has been extensively reviewed by Luisada.[1]

CASE REPORT

On 5 October 1955 a 12-year-old boy, A.A., was referred by Dr. S. J. Tsalavoutas for investigation of a possible blood dyscrasia. The family had emigrated from Greece about a year before. Thirteen days before this visit the patient noticed that his urine had become dark in colour—'the colour of tea'. He felt well and gave it no further thought. He went to school the next day but felt extremely tired and, on returning home, complained of aching pain in the left hypochondrium. His urine was still dark in colour. The mother observed that he was very pale. He went to bed and the following day was seen by the referring doctor who suspected an haemolytic episode and carried out a number of tests (Table I, 27 September).

The patient's condition rapidly improved and he returned to school. Because of the possibility of a latent blood disorder, the patient, although apparently well at this stage, was referred for further investigation.

On enquiry into the patient's history for any similar episode in the past, the following emerged. Eight years earlier, while

TABLE I

Blood	27 *September* 1955	5 *October* 1955	1 *November* 1955
Haemoglobin	10·1 g.%	12·7 g.%	15·0 g.%
RBC	3,500,000	4,220,000	
Leucocytes	5,800	7,500	
Neutrophils	56%	59%	
Eosinophils	3·0%	0·5%	
Basophils	0·5%	0·0%	
Monocytes	9·5%	4·0%	
Lymphocytes	31·0%	36·5%	
Platelets	Abundant	Abundant	
MCV		90 c.u.	
MCH		30 u.u.	
MCHC		33%	
Smear	Red cells appear normocytic and normochromic.	No target cells or spherocytes. Cells are normocytic and normochromic.	
Reticulocytes		4·1%	0·7%
Erythrocyte saline fragility		Normal	
Haemoglobin electrophoresis			Normal adult haemoglobin (A)
Thymol turbidity		Normal	
Haemoglobin and its derivatives in plasma.	Nil		
1-minute bilirubin.		0·03 mg.%	
Total bilirubin		0·20 mg.%	
Serological tests for syphilis.		Negative	
Urine			
Albumin	Trace	Nil	
Blood pigments	Negative		
Urobilinogen	+		
Urobilin	+	+	
Bilirubin	−	−	
Microscopy		Normal	

resident in Greece, the patient had an acute illness characterized by pallor and the passage of dark urine. Multiple blood transfusions were given. About 2 days before the onset of the illness the patient had eaten a few raw beans. The attending physician ascribed the illness to the beans and warned the mother of the danger to the child should he eat any in the future. The mother stated that since then she had carefully avoided giving the child these beans throughout the years. About 36 hours before the present illness he was with her when she purchased broad beans at a greengrocer's. While in the shop he ate two raw beans and about 18 hours later he partook of a meal which included a portion of the cooked beans.

The patient was an only child and the parents were not related. The mother knew of no similar illness affecting members of her family or her husband's.

Physical examination was not remarkable. Weight 95 lb, height 5 feet 2 inches. Temperature 98°F, pulse rate 80 per minute, blood pressure 120/80 mm. Hg. Neither anaemia nor jaundice was evident. Lymphadenopathy was not found. The spleen was not palpable. No abnormalities of other systems were detected.

A number of laboratory tests were performed (Table I, 5 October).

A bean was submitted to Professor J. M. Watt, Department of Pharmacology, University of the Witwatersrand, and identified as *Vicia faba*.

DISCUSSION

The diagnosis of favism was made on the basis of the history, and the identification of the bean. Although the patient had a significant anaemia (haemoglobin 10·1 g.%) direct proof that haemolysis had occurred was not established by the first tests (Table I, 27 September). About 60 hours had elapsed since the onset of the illness before the patient was seen by a doctor, and because of this delay blood pigments could not be demonstrated in the plasma or urine. This is in keeping with the experience that haemoglobinuria in favism may be present for only 1-3 days.[1] The later tests clearly show haemoglobin regeneration. Thus 8 days later (5 October) the haemoglobin was 12·7 g.%, with a reticulocytosis of 4·1%, and about 4 weeks later (1 November) the haemoglobin had risen to 15 g.% and the reticulocytes had decreased to 0·7% (Table I).

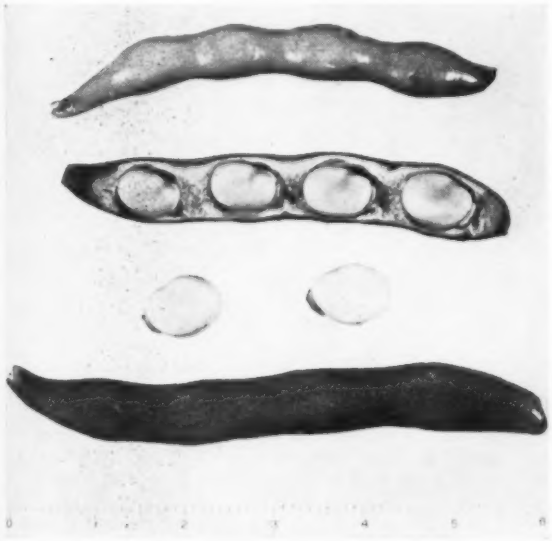

Fig. 1. The broad bean (*Vicia faba*).

The tests excluded a number of other possible causes of haemolysis. Hereditary spherocytic anaemia was ruled out by the normal range of fragility in saline. No sickling was demonstrated and electrophoresis of the haemoglobin showed it to be of normal adult type A. Neither the physical examination nor the blood smears were in keeping with Cooley's anaemia. The history and the negative serological tests for syphilis excluded paroxysmal cold haemoglobinuria.

This case of favism is reported in the hope that others may be brought to light. Cases have been reported from China,[2] but the people susceptible to the disease are largely Mediterranean in origin. Thousands of cases occur annually in Sardinia and many in Italy, Greece, Cyprus and Israel.[3] This is not merely because *Vicia faba* is an integral part of the diet of the peoples of this region; should they migrate and partake of the bean elsewhere, they will still get the disease, if susceptible. On the other hand, other races may eat the bean in Sardinia or surrounding areas and remain well. Thus the geographical location of favism is really a racial one. Furthermore, it runs in certain families in these races. There are families reported where all members have suffered from it in one form or another. Therefore the factor responsible for the disease is considered to be genetically transmitted. Wherever a susceptible population-group exists and the bean is grown, cases of favism may occur. Ten such cases have been described from the United States of America, all in people of Mediterranean origin.[4] The two cases published from England both occurred in Cypriots.[5] It has been suggested that many more cases have remained unreported.

In South Africa there is a considerable population-group, augmented by recent immigration, from countries where favism occurs in significant numbers. It is therefore of interest that broad beans are fairly widely grown throughout South Africa. Not only are they sold by greengrocers, but they are grown by individual householders, when the opportunities for the inhalation of pollen are greater than where the bean is cultivated commercially in fields distant from residential areas. The demand for broad beans stems very largely from the Mediterranean group. The flower appears from May to August and the bean is picked from July to September.[6] It is particularly during this period that cases may be expected, although the dried bean may be eaten at any time.

There appear to be as yet no specific methods whereby a diagnosis of favism can be proved. Admittedly, giving a portion of the bean would provoke a further attack but even a small quantity may precipitate a major haemolysis. The diagnosis is therefore dependant on the history.

The questioning of obscure cases of haemolytic anaemia about exposure to broad beans may well

uncover further cases. Since the prevention of potentially fatal attacks depends on avoidance of the bean it is important to make the diagnosis.

SUMMARY

A case of haemolytic anaemia due to broad beans (*Vicia faba*) in a 12-year-old Greek boy is described. To the best of our knowledge this condition has not been recorded previously in South Africa.

Broad beans are fairly widely grown throughout South Africa. It is suggested that an awareness of the condition will lead to the recognition of further cases.

Thanks are expressed to Mr. A. M. Shevitz of the Department of Medicine, University of the Witwatersrand, for the photograph.

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CLINICAL EVENING, NATAL INLAND BRANCH

A meeting of the Natal Inland Branch was held at Grey's Hospital, Pietermaritzburg, on 12 October 1955 at 8 p.m.

Dr. H. A. Kalley took the chair, and 24 members were present.

Dr. N. K. Pein described a case of a European male aged 31 years, who in December 1954 complained of loss of vision in the left eye, impotence and fatigue. The patient had acromegalic facies and examination of the fundus revealed left optic nerve atrophy. X-ray of skull showed a suprasellar tumour. This was operated on in Johannesburg and proved to be chromophobe adenoma. The right optic nerve appeared normal. Histological examination of the tumour was not possible, owing to its being excessively coagulated before excision. The patient was well for a time, but has now been readmitted complaining of failing vision of the right eye and intractable headache. There is no evidence of papilloedema. Dr. Pein then posed the questions: (1) What was the cause of the involvement of the right side? and (2) What should be done about it? Dr. J. R. Reznek suggested the use of ACTH, taking the cause to be fibrosis. Dr. A. L. Rencken suggested deep X-ray. A discussion followed, and Dr. Pein informed the meeting that the neuro-surgeons are of the opinion that re-operation is the only possible solution.

Dr. M. V. Silbert presented a case of a 24-year-old Polish male who had had his leg amputated below the knee as a result of Buerger's disease. The history was that 10 months ago this patient developed athlete's foot in both feet. The left responded to treatment, but the right failed to respond. On 28 July 1955 toes of the right foot became swollen, painful and discoloured. He was admitted to hospital on the diagnosis of fungus infection and lymphangitis. Vasodilators had no effect and the dorsalis pedis artery was not pulsating. A lumbar sympathectomy was carried out without any improvement of the circulation in the right foot. Before this a lumbar block gave a 4° rise in skin temperature of the lower leg. Exercise definitely had a bearing on the pain in the leg. Amputation of the toes was first carried out for gangrene, but this had to be followed by an amputation below the knee. Dr. Silbert stated that in his opinion this case was true Buerger's disease, according to Professor Boyd's classification, and a laboratory report on the amputated leg confirmed this diagnosis. A lively discussion followed, in which Drs. N. M. Thompson, R. L. MacKenzie, D. M. Lithgow, H. Loenstein, J. D. Woods, T. H. Whitsitt, Rencken and Pein took part.

Dr. N. M. Thompson showed X-rays of 2 cases of neonatal disease of bone. The first child was 18 days old and a twin, being the first born. The presentation was a prolapsed leg, which was oedematous, and the swelling did not subside, but persisted around the knee. There was fluid in the knee and aspiration revealed pus which grew *Staphylococcus aureus*, sensitive to all the antibiotics. The patient was treated with daily aspiration and penicillin injected locally, and is apparently normal now. The second child was 1 month old on admission, with normal temperature, a healthy appearance, a pseudo-paralysis of the right hand, and a slight enlargement of the right deltoid muscle. X-ray showed enlargement of the joint space of the right shoulder, and some new bone-formation. Aspiration drew pus, which grew non-haemolytic streptococcus (? contamination). This was treated with penicillin. These 2 patients were cases of osteomyelitis. The condition may present with green stools and diarrhoea. In treatment, bacteraemia must be controlled before the abscess is opened, to prevent the risk of metastatic abscesses.

Dr. J. F. Rivers-Moore discussed a case of a female aged 29 years with a history of sudden lower abdominal pain at 9 a.m. Bowel action relieved the pain. At 2 p.m. the pain recurred, and by 5 p.m. it was very acute, especially on the right side. No history of amenorrhoea, next period being due in 2 days' time. A clinical diagnosis of intestinal obstruction was made; an enema was returned clear, and the patient vomited. Laparotomy was performed and 1½ pints of blood removed from the abdomen. The tubes and left ovary were normal, but the right ovary had a small cavity which was oozing blood. Dr. Rivers-Moore said it would be interesting to know whether this was a primary ovarian pregnancy, or a ruptured graafian follicle, or haemorrhage into the corpus luteum. Owing to the thickness of the wall of the cavity he thought it was probably a pregnancy.

Dr. L. R. Tibbit discussed a case of a European female aged 31 years, 5-gravida, who was 3 months' pregnant and admitted as an acute abdomen. She gave a history of recurrent attacks of pain lasting 2-3 hours at a time, for the last 3 weeks, being perfectly normal in the intervals between attacks. The patient was apyrexial on admission, with a pulse of 100 per minute, a slight P.V. bleed, severe colicky pain in the lower R.I.F., and a little pale. On examination she was very tender in the R.I.F., and pressure anywhere in the abdomen gave pain in this area. A laparotomy revealed 1 pint of pure blood in the abdominal cavity. The right tube showed a thickening half-way along, and fresh blood was issuing from the fimbriated end of the tube. Salpingectomy was performed, which showed a tubal pregnancy which had been dead for a considerable time. The patient has made an uneventful recovery and a uterine pregnancy is continuing.

Dr. W. Fabian discussed a case of diphtheria in a child aged 3¼ years. The child had been sick for 4 days before being seen by a doctor. The diagnosis was confirmed by a throat swab. The patient recovered uneventfully after 5½ weeks. Fourteen months later the child complained of a sore throat and was clinically again diphtheria, but owing to the previous attack Dr. Fabian was hesitant to make the diagnosis. However, a throat swab confirmed the second attack as diphtheria, and the child was much more seriously ill than on the previous occasion. An interesting discussion followed embracing the effects of immunity conferred by one attack and the merits of immunization. This child had not been immunized.

Dr. J. R. Reznek presented the meeting with a most enjoyable colour ciné-film of a case of hypernephroma. He was congratulated on the excellence of this, his first attempt at a medical film. The sequences of the film covered the case history, the laboratory findings, cystoscopy and the operation. The details revealed in the operation made it perfectly easy for the audience to imagine they were watching the actual operation. Dr. Reznek gave a commentary throughout the showing of the film. The Branch recorded their hearty congratulations.

The meeting closed at 10.45 p.m.

NEW FACULTY OF MEDICINE : UNIVERSITY OF STELLENBOSCH

The following appointments at the Stellenbosch Medical School have been announced:

Dean of the Faculty of Medicine and Professor of Surgery. Mr. F. D. du T. van Zyl, M.B., Ch.M. (Cape Town), F.R.C.S. (Edin.). Mr. van Zyl is Surgeon to the Groote Schuur Hospital, Cape Town, and senior lecturer in Surgery at the University of Cape Town, where he has several times served as Acting Professor of Surgery. Mr. van Zyl will for the present carry on with his work at Groote Schuur Hospital and the Cape Town Medical School, and will continue in his private surgical practice in Cape Town.

Professor of Medicine. Dr. A. J. Brink, M.D. (Pretoria), M.B., Ch.B. (Rand), M.R.C.P. (Edin.), of the Pretoria Hospital and the Department of Internal Medicine, University of Pretoria.

Professor of Obstetrics and Gynaecology. Dr. J. N. de Villiers, M.B., Ch.B., M.O. & G. (Cape Town), who was Registrar in the Division of Obstetrics and Gynaecology, Cape Provincial Administration and the University of Cape Town, attached to Groote Schuur and other hospitals, and who at present is working at the Hammersmith Postgraduate Hospital, London, under a Nuffield Travelling Fellowship.

Senior Lecturer and Head of the Department of Anatomy. Dr. T. Coetzee, M.B., M.Ch. (Cape Town) who is at present Lecturer in Anatomy at the Durban Medical School, University of Natal.

Dr. Coetzee will assume duty in 1956 and Professors van Zyl, Brink and de Villiers in 1958.

The Cape Provincial Administration has appointed Dr. J. H. Cairns, M.B., Ch.B. (Cape Town) as Medical Superintendent of the projected New Parow Hospital, which is eventually to serve as the teaching hospital of the Stellenbosch Medical School. Dr. Cairns has for the past 2 years been professional assistant to the Director of Hospital Services, Cape Province, and previously served as Medical Superintendent, Provincial Hospital, Port Elizabeth. He is to visit Europe and America to investigate hospital construction, equipment and administration.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Koppel Furman, B.Sc., M.B., B.Ch., M.R.C.P. (Edin.), has commenced practice as a specialist physician at 215 Lister Building, Johannesburg. Telephone: rooms 23-6038, residence 42-7761.

* * *

Experimental Research into Problems of Ageing. Candidates wishing to submit entries for the 1955-56 Ciba Foundation Awards of papers descriptive of research relevant to basic problems of ageing are reminded that these must reach the Ciba Foundation not later than 10 *February* 1956.

Information about the Awards, for those not already aware of the conditions, may be obtained on application from Dr. G. E. W. Wolstenholme, O.B.E., Director and Secretary to the Executive Council, 41 Portland Place, London, W. 1.

* * *

Dr. L. J. te Groen, F.R.C.O.G., is starting practice as a specialist obstetrician and gynaecologist at 718 Medical Centre, Pretorius Street, Pretoria, from 3 January 1956.

* * *

Dr. L. J. te Groen, F.R.C.O.G. begin vanaf 3 Januarie 1956 as spesialis in obstetrie en ginekologie te praktiseer te Mediese Sentrum, 718 Pretoriusstraat, Pretoria.

CORRESPONDENCE : BRIEWERUBRIEK

CEREBRAL PALSY

To the Editor: The attention of my National Council has been directed to a letter[1] which appeared in the issue of your *Journal* of 8 October 1955. It is felt that the impression to be derived from that article is somewhat inadequate, and in some respects open to contradiction. In these circumstances my Council has issued the following statement to which it is trusted you will grant the courtesy of your columns:

'Cerebral Palsy, until comparatively recent years considered a condition beyond the scope of medical aid, has latterly been recognized as worthy of particular investigation. Spontaneously, in America, the United Kingdom and certain countries of Europe, a new awareness of habilitative potentialities became apparent. Inspired largely by determined parents, limited research was undertaken by recognized medical authorities, whose observations led to the establishment of diagnostic units which experimented with various types of equipment and therapeutic techniques.

'The development since those modest beginnings has been remarkable. Notwithstanding that cerebral palsy is a highly complex social, educational, psychological and economic problem, what may now be regarded as an adequate basis for the habilitation of the majority of sufferers has emerged.

'In South Africa similar development took place with individual parents privately combining to establish facilities for the education and social adjustment of their affected children. These "schools" grew to include provision for physiotherapy, speech training and, naturally, occupational therapy. The approach at this stage was necessarily educational with the various therapies incidental and ancillary. Expert medical direction was at that time lacking.

'Newly labelled "a neuro-psychological problem with orthopaedic involvement", cerebral palsy is seen to require a high degree of medical intervention. The need for accurate diagnosis with an individual pattern of therapy calls for a "diagnostic clinic", adequately equipped and staffed, as the first stage toward habilitation. Here provision is made for effective screening, the elimination of pseudo-CPs and the instruction of parents.

'To assess the standard of ability in every function is a vital prerequisite to treatment in any form. The process involves a series of medical examinations, and observation by trained therapists, over periods which will vary with individual cases. Dr. Bronson Crothers—a noted international authority—has warned that precipitate treatment, such as physiotherapy, for example, may do more harm than good.

'Armed with this basic picture of a CP's capability, it is possible to devise a pattern of interim activities within his reach, a process which constitutes "Stage 1" on the road to habilitation. Meanwhile steps are taken to provide obvious aids, such as braces, crutches etc., which may be indicated. Medication is frequently necessary and all these primary requirements should receive attention before any deliberate therapy is undertaken.

' "Stage 2" is initiated by unobtrusive development of latent functions. Play is the background for all forms of therapy, and the patient is encouraged by example and incentive to strive for new skills—modest when considered individually, but having a cumulative effect of great value.

'As soon as practicable newly acquired control is directed toward purposeful activities. "Stage 3" seeks gradual improvement in the fundamental arts of feeding, walking, talking and dressing, whilst care is taken to encourage any other ability that may be present.

'It is the work of a "diagnostic and treatment centre" to carry out the functions described above, and it was with these facts in mind that the Southern Suburbs Clinic of the United Cerebral Palsy Association of South Africa was conceived.

'Having completed a programme of therapy as envisaged above, a considerable number of CPs may proceed to "special schools" where modified curricula and sheltered conditions will facilitate their academic progress. Here, too, CP's with moderate involvement could further their education whilst receiving therapeutic assistance either on the premises or extramurally.

'Mildly affected CPs and those sufficiently habilitated should be admitted to normal schools as soon as possible. It is not advisable to segregate the cerebral palsied. They should, on the contrary, be integrated into the community. No better route to a human pattern of living could be devised than early acceptance by normal boys and girls. Antisocial or defeatist tendencies, otherwise commonly encountered, are checked, and habilitation is sub-

stantially advanced by a continuing process of accomplishment and progress'.

In their letter[1] your correspondents state: 'In addition we wish to point out that, since the Forest Town School started in 1948, other cerebral palsy schools have been established—in Pretoria in 1950 and in Cape Town in 1954. *These are the only recognized cerebral palsy schools and treatment centres in South Africa*' (my italics).

My council particularly regrets the suggestion implied in the sentence in italics that the Southern Suburbs Treatment Centre of the United Cerebral Palsy Association of South Africa is not 'recognized'. This might be construed to imply illegality—a serious and wholly unfounded allegation. The fact is that, unlike the schools mentioned, 'diagnostic clinics' do not qualify for educational grants. It is hoped that State financial assistance will eventually be made available by the Department of Health. Meanwhile, all the expenses of the existing U.C.P.A. European clinic at Rosettenville—as well as the proposed non-European centre to be opened early next year—are being provided by public support exclusively.

The United Cerebral Palsy Association of South Africa is a national welfare organization registered in terms of Sec. 9 of the Welfare Organizations Act 1947, and is therefore fully 'recognized' by the State and public at large.

H. Kessler
National Chairman
United Cerebral Palsy Association
of South Africa
P.O. Box 8665
Johannesburg
30 November 1955

1. Vorwerg, F. M. T. and Levitt, S. (1955): S. Afr. Med. J., **29**, 976.

MONDELINGE MANIFESTASIES VAN GESTELSIEKTES

Aan die Redakteur: Ek het dr. H. Goldin se artikel oor hierdie onderwerp met genot gelees.

In verband met die sogenaamde dispeptise nekus het ek 'n ondervinding gehad wat moontlik kollegas tot hulp mag wees:

'n Ongetroude onderwyseres het gereeld elke maand gedurende haar maandstonde een of meer klein sere in die mond gekry. Soms het sy dit tussentyds ook gekry. Sy ly bowendien aan premenstruele spanning en dismenorree. 'n Seer is 2 tot 4 millimetres in deursnee; dit is wit en omring met 'n rooi ringetjie.

Greene en Dalton (*British Medical Journal*, 9 Mei 1953) noem mondsere as een van die moontlike uitings van premenstruele spanning en om dié rede het ek hulle behandeling van progesterone in die laaste helfte van die siklus toegepas. Dit was net soos plaaslike en parenterale antibiotika, heeltemal sonder sukses. Vitamiene- en breësprektum antibiotiese behandeling deur 'n internis was ook sonder sukses.

Gedurende 'n griepaanval het die mondsere herhaal en het die gedagte ontstaan dat medisyne die oorsaak kon wees. Aspirien, in al sy vermommings, is belet en die pasiënt het in die afgelope 7 maande geen mondsere gehad nie.

'n Ander pasiënt wie se mondsere ons nie kon genees nie, het met aspirien-onthouding ook herstel. Die mak honde byt die seerste!

B. P. J. F. Coetzee
Dealesville, O.V.S.
14 Desember 1955

1. Goldin, H. (1955): S. Afr. T. Geneesk., **29**, 1159.

THE OUT-PATIENT MANAGEMENT OF ACUTE LUNG ABSCESS

To the Editor: May I thank Dr. Crawshaw[1] for his interest and kind criticism of my article[2] on this subject.

My reference to the difficulty in getting a 'true history' was directed to the association of alcoholism particularly. Allowing for the inaccuracies in the history-taking in Natives, I still maintain that clubbing of the fingers is an early sign in acute lung abscess, being present as early as 2-3 weeks from the commencement of symptoms. This view is supported by observation of a larger series of cases than that presented in my article and aided by the facilities at this clinic for picking up chest lesions at an early stage.

Unfortunately the X-ray plates did not reproduce well and I do not blame Dr. Crawshaw for not accepting the final plate as evidence of cure. Although the X-ray film does show minimal fibrosis and this stage is labelled 'radiological cure', I also did not accept this as a cure for the majority of the patients, as they were still left with the virtual cause of the lung abscess, viz. oral sepsis. I have not found it possible to persuade the Native patients to have their teeth removed when dentures are so difficult to come by. I had merely coped with a complication of oral sepsis, viz. lung abscess, and I expect these cases to have recurrences while oral sepsis continues, as would be likely to happen had the lung abscess been dealt with surgically. The term 'radiological cure' was not a happy one and was intended to stress the fact that symptoms disappeared when radiological appearance still suggested pathological activity ('symptomatic cure') and that this occurred days to weeks before scarring on X-ray was firm and minimal. The moral is to press on with treatment to this stage and not be guided by symptoms. I do not consider that bronchography is of particular use in the assessment of 'cure', which embraces other factors than X-ray appearance alone. I would expect distortion of bronchi or non-filling in the area of scarred lung; thus I regard this as a normal result of healing and I do not expect this bronchiectasis *per se* to be a cause of flare-up, except in the lower zones where the bronchi are dependent. These areas of the lung have been pointed out as not responding well to medical treatment alone when infected with lung abscess, or tubercle for that matter, and are not out-patient propositions.

Dr. Crawshaw quite correctly challenges the statement that before the advent of antibiotics 'fibrosis was an intended end-result of infection'. This was an error on my part in proof-reading. 'Treatment' should replace 'infection'. I had in mind the fibrosis of healed tubercle and the fibrosis-stimulating measures of iodine packs in the preliminary-stage operation in lung abscess intended to localize the abscess and aid exteriorization for subsequent drainage.

With reference to the question of physical activity in anti-microbial therapy, I agree that good results are obtained with conventional bed rest. The object of my paper was to point out that equally good results were found in certain types of lung abscess treated as out-patients, and certain theories were suggested to explain factors in out-patient treatment which were apparently beneficial to healing and which limited fibrosis. Rest favours fibrosis and the protection of organisms by fibrosis. Activity hinders fibrosis and favours the invasiveness of organisms and the spread of disease. This is seen in tubercle, a disease which offers a 'slow motion' observation of a lung infection. Physical activity has a cortisone-like inflammatory spreading effect. It has been shown *in vitro* that the tubercle bacillus in an active and dividing state is more sensitive to streptomycin and isoniazid than in a resting phase. In a similar way I believe that physical activity of an optimum degree renders the organisms more accessible to the drugs in a condition like lung abscess.

Dr. Crawshaw cannot see why the maintenance of good muscular tone through ambulation, as compared to the atrophy of bed rest, should favour healing of a lung abscess. I consider it accepted teaching that in any infection attention must be given to general measures for improving health as well as to local measures. Apart from any other physiological considerations I feel that the maintenance of morale alone must favour healing. I notice that Dr. Crawshaw includes physiotherapy in his regime of treating lung abscess. Could he not regard ambulation as physiotherapy self-applied?

Finally, I have no intention of 'starting a Union-wide campaign for the treatment of acute lung-abscess in out-patient departments'. I stressed the seriousness of the condition and its complications, but wished to demonstrate to colleagues that *certain types* of acute lung-abscess can be managed in the out-patient department provided X-ray facilities are available. The inference may also be drawn that where such a case is hospitalized, a reasonable degree of ambulation would favour drainage and so promote healing and limit fibrosis.

H. Dubovsky
Durban Chest Clinic
Warwick Avenue
Durban
15 December, 1955.

1. Crawshaw, G. R. (1955): S. Afr. Med. J., **29**, 1184 (10 December).
2. Dubovsky, H. (1955): *Ibid.*, **29**, 1097 (19 November).

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CONTENTS — INHOUD

Die Mediese Vereniging van Suid-Afrika
The Medical Association of South Africa

AGENTSKAP-AFDELING : AGENCY DEPARTMENT

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* * *

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(944) EAST RAND. Locum as from 21 January till 6 February 1956. Practically no night work. Partnership practice. Terms to be arranged.

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(942) LARGE TRANSVAAL HOSPITAL TOWN. A locum to start as soon as convenient for a period up to 3 months or longer. General practice, plus surgery. Hospital appointment. Salary £3 3s. 0d. per day, plus all found.

(941) JOHANNESBURG. Locum for February. Preferably someone with experience of private practice and contract practice. £3 3s. 0d. per day, plus £10 p.m. car allowance, plus petrol and oil and board and lodging.

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(930) NEAR JOHANNESBURG. Locum as from 1 tot 31 January 1956. Preferably someone with own car. Salary £3 3s. 0d. per day, plus all found.

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In established suite of Rooms, the following is available for immediate occupation: Consulting room with separate interleading examination room, plus sharing of fully equipped European and non-European waiting rooms, and office: and the services of book-keeper receptionist (trained sister). Apply 401 Ingrams' Corner, Hillbrow, Johannesburg.

IMPORTANT NOTICE

The Federal Council wishes all medical practitioners, who intend applying for contract practice appointments advertised in these columns, to consult the Honorary Secretary of the Branch of the Medical Association of South Africa in whose area such an appointment falls in order that they may know whether the terms of the appointment are satisfactory. Contract practice appointments include all those made to Medical Benefit Societies, Sick Funds and Factories. The addresses of the Honorary Secretaries of Branches are as follows:

Border: Dr. L. L. Alexander, P.O. Box 335, East London.
Cape Eastern: Dr. D. A. H. Johnson, 120, High Street, Grahamstown.
Cape Midland: Dr. P. Jabkovitz, 1, Glenairlie, 69, Cape Road, Port Elizabeth.
Cape Western: Dr. P. C. W. Madden, P.O. Box 643, Cape Town.
East Rand: Dr. W. M. Bezuidenhout, P.O. Box 536, Benoni.
Griqualand West: Mr. A. B. de Villiers Minnaar, Dutoitspan Road, Kimberley.
Natal Coastal: Dr. N. R. Pooler, 112, Medical Centre, Field Street, Durban.
Natal Inland: Dr. T. H. Whitsitt, P.O. Box 285, Pietermaritzburg.
Northern Transvaal: Dr. E. Fasser, 28, Administrative Building, General Hospital, Pretoria.
O.F.S. and Basutoland: Dr. C. V. van der Merwe, P.O. Box 834, Bloemfontein.
Southern Transvaal: Dr. M. Peskin, Medical House, 5, Esselen Street, Johannesburg.
South West Africa: Dr. H. C. Paradisgarten, P.O. Box 1667, Windhoek.
Transkei: Dr. J. H. Hofmeyr, P.O. Box 27, Umtata.

O.V.S. Provinsiale Hospitaal

BETHLEHEM

VAKATURE : DEELTYDSE GENEESKUNDIGE EN CHIRURGIESE PERSONEEL

Aansoeke van behoorlik gekwalifiseerde en geregistreerde Geneeshere word ingewag vir die betrekking van Deeltydse Geneesheer by die Provinsiale Hospitaal, Bethlehem vir die tydperk 1 Februarie 1956 tot 31 Maart 1957.

Aansoeke met volle besonderhede insake vorige ondervinding, kwalifikasies, tesame met gewaarmerkte afskrifte van getuigskrifte en geboortesertifikate moet gerig word op die voorgeskrewe Vorm Z. 83, verkrygbaar van die Sekretaris, Provinsiale Hospitaal, Bethlehem of by enige Magistraatskantoor, en moet die ondergetekende nie later as 18 Januarie 1956 bereik nie.

J. P. A. Venter
Geneesheer-Direkteur
N 133050

Bethlehem
3 Desember 1955

O.F.S. Provincial Hospital

BETHLEHEM

VACANCY : PART-TIME MEDICAL SURGICAL STAFF

Applications are invited from duly qualified and registered Medical Practitioners for a vacancy on the Part-time Staff of the Provincial Hospital, Bethlehem for the period 1 February 1956 to 31 March 1957.

Applications on prescribed Forms Z. 83, obtainable from the Secretary, Provincial Hospital, Bethlehem, or any Magistrate's office, giving full particulars of previous experience and accompanied by certified copies of testimonials and birth certificates, must reach the undersigned on or before 18 January 1956.

J. P. A. Venter
Medical Superintendent
133050

Bethlehem
3 December 1955

Provincial Administration of the Cape of Good Hope

UNIVERSITY OF CAPE TOWN : JOINT MEDICAL STAFF FOR GROOTE SCHUUR AND OTHER TEACHING HOSPITALS : VACANCY

Applications are invited from registered Medical Practitioners (Registered Specialists) for appointment to the following post:

Department of Obstetrics and Gynaecology (Part-time).

Medical Practitioner, Grade G, and possible consequential vacancies for Medical Practitioner, Grades F and E.

Remuneration

Grade G—£196 per annum per session.
Grade F—£175 per annum per session.
Grade E—£164 per annum per session.

The conditions of service are governed by the relevant Ordinance and Regulations as well as the agreement entered into between the Provincial Administration and the University of Cape Town.

The Joint Medical Staff is required to serve jointly the Provincial Administration and the University of Cape Town.

A session shall be 4 hours per week, not necessarily continuous and/or clinical and teaching work.

Candidates for the posts of Medical Practitioner, Grades G, F and E, must have not less than three years' experience after registration as a Specialist in Obstetrics and Gynaecology.

Candidates must state the number of sessions they are prepared to undertake.

Application must be made in duplicate on the prescribed form, Staff 23, which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from any Provincial Hospital or School Board office in the Cape Province.

The completed application forms must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 21 January 1956.

N 61441

UNIVERSITY OF CAPE TOWN

SENIOR LECTURER IN PHYSIOLOGY

Applications are invited for the post of senior lecturer in physiology. The successful candidate will take part in the teaching of medical and science students and will be in charge of either the experimental or the histological laboratory. A medical qualification is desirable.

The salary scale is £1,200×50—1,450 per annum plus a temporary cost of living allowance for a married man (at present £234 per annum). A higher initial salary may be given on the grounds of qualifications and experience.

Applications (with copies of testimonials) should state age, qualifications, experience, publications and research interests, and should give the names of two referees whom the University may consult. Memoranda giving the general conditions of appointment and information on the work of the department should be obtained from the Registrar, University of Cape Town, Private Bag, Rondebosch, to whom applications must be submitted not later than 16 February 1956. The University reserves the right to appoint a person other than one of the applicants or to make no appointment.

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Vakante Deeltydse Distriksgeneesheerskappe

Aansoeke om ondergenoemde poste van distriksgeneeshere, met vermelding van land van geborte, kwalifikasies, ondervinding vorige en teenswoordige betrekkings en die vroegste datum waarop diens aanvaar kan word, indien aangestel, word deur die Sekretaris van Gesondheid, Posbus 386, Pretoria, ingewag en moet hom voor of op 11 Januarie 1956, bereik. Afskrifte van getuigskrifte mag gestuur word.

Invloedwerwing deur of ten behoewe van 'n applikant stel hom bloot aan diskwalifikasie.

Die aanstelling is deeltyds en privaat praktyk word toegelaat.

Applikante moet ook vermeld of hulle albei amptelike tale kan praat, lees en skryf, asook of hulle melaatsheid en veneriese siekte kan diagnoseer.

Applikante moet verder vermeld of hulle ondervinding as 'n geneeskundige gesondheidsbeampte of in 'n soortgelyke hoedanigheid het. As om meer as een pos aansoek gedoen word, moet 'n afsonderlike aansoek ten opsigte van elkeen ingedien word.

Plek	*Salaris per Jaar*	*Toelaag vir Medisyne per Jaar*
Kaapprovinsie:	£	£
Alicedale	100	20
Fort Beaufort	200	45
Marydale	300	20
Pearston	200	25
Pofadder	370	40
Rhodes	350	25
Ugie	180	20
Indwe	175	20
Transvaal:		
Belfast	250	24
Janseput	350	25
Oranje-Vrystaat:		
Witzieshoek	250	20

Die salaris dek alle gewone en roetine dienste, dog reistoelae teen 1s. per myl vir alle afstande wat buite 'n omtrek vir die myl vanaf die standplaas afgelê word, nagverblyf teen 15s. en bykomende vergoeding vir sekere dienste word betaal, asook gelde vir bywoning van hofsittings en geregtelike lykskouings ooreenkomstig die skaal van die Departement van Justisie.

Aansoekvorms en kopieë van kontrakvorms word op aansoek verskaf.

3669

Stadsgebied van Estcourt

(Kennisgewing Nr. 76/1955)

VAKATURE MEDIESEGESONDHEIDSBEAMPTE (DEELTYDS)

Aansoeke word ingewag vir bovermelde pos teen remunerasie van £159 14s. 0d. per jaar plus duurtetoeslag van £80 6s. 0d. per jaar plus 'n fooi van £120 per jaar vir die ondersoek van Naturelle (ex Arbeidsburo) plus 'n fooi van £120 per jaar vir Kliniekdienste.

Afskrifte van die voorwaardes aan die pos verbonde is op aansoek verkrygbaar. Volledige besonderhede ten opsigte van professionele kwalifikasies. Kennis van beide amptelike landstale ens. moet verstrek word.

Aansoeke sluit 12 uur middag, Saterdag, 7 Januarie 1956.

Robus Menne
Stadsklerk

Stadskantoor
Estcourt
20 Desember 1955

Vacant Part-Time District Surgeoncies

Applications for the undermentioned district surgeoncies accompanied by full particulars as to date and country of birth, qualifications, experience, previous and present appointments of the applicants and the earliest date on which they can assume duty, if appointed, should reach the Secretary for Health, P.O. Box 386, Pretoria, not later than 11 January 1956. Copies of testimonials may be submitted.

Canvassing by or on behalf of any applicant is liable to disqualify him.

The appointments are on a part-time basis and private practice is not precluded.

Applicants should state whether they can speak, read and write both official languages, also whether they are competent to diagnose leprosy and venereal disease.

Applicants should also state whether they have any experience as a Medical Officer of Health or in any similar capacity. If more than one post is applied for, a separate application should be submitted in respect of each.

Place	*Salary per Annum*	*Drug Allowance per Annum*
Cape Province:	£	£
Alicedale	100	20
Fort Beaufort	200	45
Marydale	300	20
Pearston	200	25
Pofadder	370	40
Rhodes	350	25
Ugie	180	20
Indwe	175	20
Transvaal:		
Janseput	350	25
Belfast	250	24
Orange Free State:		
Witzieshoek	250	20

The salaries cover all ordinary and routine services but travelling allowances of 1s. per mile for all mileage travelled outside a radius of three miles from headquarters, night detention at 15s. and supplementary fees for certain other services will be payable, also fees for attendance at courts and inquests in accordance with the tariff of the Department of Justice.

Forms of application and copies of draft agreement will be furnished on application.

3669

Borough of Estcourt

(Notice No. 76/1955)

VACANCY PART-TIME MEDICAL OFFICER OF HEALTH

Applications are invited for the above position at a remuneration of £159 14s. 0d. per annum plus cost of living allowance at £80 6s. per annum for the examination of Natives (Ex Labour Bureau) plus a fee of £120 per annum in respect of Clinic Duties. Copies of conditions attached to the position are obtainable on request. Full details of professional qualifications, knowledge of both Official Languages etc. to be furnished.

Applications close on 7 January 1956 at 12 noon.

Robus Menne
Town Clerk

Civic Building
Estcourt
20 December 1955

Vakatures vir Besoekende Narkotiseurs en Borskas-Chirurg (Deeltyds)

RIETFONTEINHOSPITAAL, JOHANNESBURG

Aansoeke om aanstelling in ondergenoemde betrekkings in die personeel van die Rietfonteinhospitaal, Johannesburg, word van behoorlik gekwalifiseerde kandidate ingewag:

Betrekking	*Besoldiging aan betrekking verbonde*	*Pligte*
Narkotiseur (2) (deeltyds)	£300 per jaar (insluitend)	Een operasiesessie per week.
Borskas-chirurg (2) (deeltyds)	£300 per jaar (insluitend)	Een operasiesessie per week.

Kandidate moet Suid-Afrikaanse burgers of burgers van 'n Statebondsland of die Republiek Ierland en tweetalig wees en moet minstens drie jaar in die Unie van Suid-Afrika of Suidwes-Afrika gewoon het.

Registrasie by die Suid-Afrikaanse Mediese en Tandheelkundige Raad as 'n spesialis in die besondere spesialiteit is 'n noodsaaklike vereiste vir aanstelling in enigeen van die betrekkings.

Van die aangestelde persone sal verwag word om saam te werk in alle navorsing wat met hulle spesialiteite in verband staan en om, waar moontlik, personeelsamesprekings by te woon. Hulle sal vir hulle eie vervoer verantwoordelik wees en die koste verbonde daaraan self moet dra.

Nadere besonderhede in verband met hierdie voorgenome aanstellings is van die Mediese Superintendent van die betrokke hospitaal verkrygbaar.

Daar moet aansoek gedoen word op die voorgeskrewe vorms (Z. 83 en S.D.K. 8 (*a*)) wat van die Sekretaris van Gesondheid, Posbus 386, Pretoria, verkrygbaar is.

Die sluitingsdatum vir die ontvangs van die aansoeke is 7 Januarie 1956.

3751

Siektefonds van die Suid-Afrikaanse Spoorweë en Hawens

Aansoeke word ingewag van geregistreerde mediese praktisyns vir aanstelling in die ondervermelde betrekkings:

1. Spoorwegdokter, Bellair: Salaris £979 per jaar.
2. Spoorwegdokter, Fynnlands: Salaris £694 per jaar.
3. Spoorwegdokter, Pietermaritzburg 'C': Salaris £1,412 per jaar.
4. Spoorwegdokter, Pietermaritzburg 'D': Salaris £369 per jaar.

Volle besonderhede in verband met die aanstellings kan verkry word van en aansoeke gerig word aan:

Die Distriksekretaris,
Natal Distriksiekefondsraad,
Belgravegebou,
Smithstraat,
Durban.

L.W.—1. Die aanstelling word op 'n deeltydse grondslag gemaak en behalwe die betrokke salarisse is die gelde en toelaes waarvoor die Siekefonds Regulasies voorsiening maak, ook betaalbaar.

2. Werwing deur of ten behoewe van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Sluitingsdatum vir aansoeke: 21 Januarie 1956.

P. J. Klem
Hoofsekretaris

Johannesburg
31 Desember 1955

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Vacancies for Visiting Anaesthetists and Thoracic Surgeons (Part-time)

RIETFONTEIN HOSPITAL, JOHANNESBURG

Applications are invited from suitably qualified candidates for appointment to the undermentioned situations which exist on the establishment of the Rietfontein Hospital, Johannesburg:

Situation	*Remuneration attaching to Situation*	*Duties*
Anaesthetist (2) (Part-time)	£300 per annum (inclusive)	One operation session per week.
Thoracic Surgeon (2) (Part-time)	£300 per annum (inclusive)	One operation session per week.

Candidates must be South African citizens, or citizens of a Commonwealth country or citizens of the Republic of Ireland, be bilingual and have resided in the Union of South Africa or in South West Africa for at least three years.

Registration with the South African Medical and Dental Council as a specialist in the particular speciality is an essential requirement for appointment to the situations.

The appointees will be expected to cooperate in any research work connected with their specialities and to attend staff consultations when possible. They will be responsible for their own transport arrangements and at their cost.

Further information in regard to these proposed appointments can be obtained from the Medical Superintendent of the hospital in question.

Application must be made on the prescribed forms (Z. 83 and P.S.C. 8 (*a*)) which are obtainable from the Secretary for Health, P.O. Box 386, Pretoria.

The closing date for receipt of applications will be 7 January 1956.

3751

Departement van Mynwese

AANSTELLING VAN DEELTYDSE SPESIALIS IN DIE MEDIESE SILIKOSEBURO

Aansoeke word ingewag van geregistreerde Borschirurge om aanstelling in die Mediese Silikoseburo, Johannesburg, in die hoedanigheid van deeltydse diagnostikus teen 'n salaris bereken op die volgende basis:

6 uur per week	£660 per jaar.
9 uur per week	£990 per jaar.
12 uur per week	£1,320 per jaar.
15 uur per week	£1,650 per jaar.
18 uur per week	£1,980 per jaar.

Die aanstelling geskied op kontrak vir 'n tydperk van 3 jaar, wedersydes opsegbaar met drie maande kennisgewing.

Applikante moet besonderhede van hulle kwalifikasies ouderdom en vorige ondervinding verstrek en hul aansoeke instuur on die Sekretaris van Mynwese, Privaatsak 59, Pretoria, voor of op 14 Januarie 1956, te bereik.

M.M. Staf 1/32/2
3549

VENNOOTSKAP

Vennootskap aangebied as uitbreiding in bestaande twee-man-vennootskap. Plattelandse dorp in Transvaal met moderne hospitaal en aangeneme werk.

Aanstellings aan praktyk verbonde maar daar is min rywerk. Drie tot ses maande proef tydperk. Meld snykunde ondervinding.

Voorwaardes ens. sal gereel word verkieslik met onderhoud. Skryf aan A.B.B., Posbus 643, Kaapstad.

Printed by National Commercial Printers, Elsies River, and Published by the Proprietors, *The Medical Association of South Africa*, Medical House, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'.

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Protamine Zinc Insulin, 40 *units per c.c., in bottles of* 5 *and* 10 *c.c., 80 units per c.c., in bottles of* 5 *c.c.*

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Insulin Zinc Suspension (Crystalline)—Ultralente, 40 *units per c.c., in bottles of* 10 *c.c.*

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